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No.

Boston
Medical Library,
19 BOYLSTON PLACE.

TRANSACTIONS

OF THE

PATHOLOGICAL SOCIETY

OF PHILADELPHIA.

VOLUME I.—New Series.
VOLUME XIX.—Old Series.

CONTAINING THE REPORT OF THE PROCEEDINGS FROM OCTOBER, 1897, TO JUNE, 1898.

EDITED BY
ALONZO E. TAYLOR, M.D.,
RECORDER OF THE SOCIETY.

PHILADELPHIA:
PRINTED FOR THE SOCIETY BY THE AVIL PRINTING COMPANY.
1898.

BOSTON MEDICAL
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E. H. B.

NOTIFICATION is hereby given that beginning with November, 1897, the proceedings of the Philadelphia Pathological Society will be published monthly, the issues appearing on the 15th of each month from November to June, inclusive. These issues constitute the sole publications and scientific records of the Society, and no bound volume of the transactions will be issued hereafter, except Volume XVIII, comprising the proceedings of 1895-96 and 1896-97, which will appear early in 1898.

Owing to a printer's error, the paging of issue No. 2, December, 1897, was 1 to 20, whereas it should have been 21 to 40, inclusive; the issue No. 3, January, 1898, being pages 41 to 64.

Erratum.

Readers will please notice that in No. 2 of the transactions the pages should be numbered from 21 to 40 inclusive, instead of from 1 to 20. In indexing their contents the pages have been given their proper numbers.

FORMER PRESIDENTS.

SAMUEL D. GROSS, M. D., LL. D., D. C. L. OXON., LL. D., CANTAB., elected 1857.

RENE LA ROCHE, M. D., elected 1858.

ALFRED STILLE, M. D., LL. D., elected 1859, '61, and '62.

EDWARD HARTSHORNE, M. D., elected 1860 and '63.

J. M. DA COSTA, M. D., LL. D., elected 1864, '65, and '66.

JOHN H. PACKARD, M. D., elected 1867 and '68.

S. WEIR MITCHELL, M. D., LL. D., elected 1869.

JOHN ASHHURST, JR., M. D., LL. D., elected 1870.

JAMES H. HUTCHINSON, M. D., elected 1871 and '72.

WILLIAM PEPPER, M. D., LL. D., elected 1873.

H. LENOX HODGE, M. D., elected 1876.

S. W. GROSS, M. D., elected 1879.

JAMES TYSON, M. D., elected 1882 and '83.

E. O. SHAKESPEARE, M. D., elected 1884.

J. C. WILSON, M. D., elected 1885 and '86.

F. P. HENRY, M. D., elected 1887 and '88.

HENRY F. FORMAD, M. D., elected 1889 and '90.

ARTHUR V. MEIGS, M. D., elected 1891 and '92.

J. H. MUSSER, M. D., elected 1893, '94, '95, and '96.

OFFICERS AND COMMITTEES
OF THE
PATHOLOGICAL SOCIETY OF PHILADELPHIA.

(Elected at the Annual Meeting, October 14, 1897.)

PRESIDENT.

W. E. HUGHES, M. D.

VICE-PRESIDENTS.

F. A. PACKARD, M. D., A. STENGEL, M. D.,
C. W. BURR, M. D., J. GUITÉRAS, M. D.

SECRETARY.

D. RIESMAN, M. D.

TREASURER.

T. S. WESTCOTT, M. D.

RECORDER.

A. E. TAYLOR, M. D.

CURATOR.

J. D. STEELE, M. D.

BUSINESS COMMITTEE.

A. C. ABBOTT, M. D., F. A. PACKARD, M. D.,
J. M. SWAN, M. D., J. SAILER, M. D.,
J. A. SCOTT, M. D.

COMMITTEE ON MORBID GROWTHS.

D. RIESMAN, M. D., J. McFARLAND, M. D.,
A. O. J. KELLY, M. D.

COMMITTEE ON PUBLICATION.

A. STENGEL, M. D., F. P. HENRY, M. D.,
A. A. ESHNER, M. D.

LIST OF MEMBERS.

Members marked O. M. are those originating the Society.

ELECTED

1894 Abbott, A. C., 221 South Forty-fourth Street.
1887 Allyn, Herman B., S. E. Corner Forty-second Street and Baltimore Avenue.
1889 Anders, J. M., 1603 Walnut Street.
1894 Arnold, John P., 3722 Walnut Street.
1861 Ashhurst, John, Jr., 2000 DeLancey Place.
1891 Ashton, Thomas G., 1533 Pine Street.
1895 Ashton, William E., 2011 Walnut Street.

1897 Babcock, W. Wayne, 2133 North Front Street.
1898 Baggs, Albert N., Abingdon, Pa.
1887 Baker, G. F., 1818 Spruce Street.
1888 Balliet, Tilghman M., 3705 Powelton Avenue.
1883 Barton, Isaac, 111 North Sixteenth Street.
1885 Beates, Henry, Jr., 1504 Walnut Street.
1871 Bennett, Wm. H., 2105 Spruce Street.
1887 Berens, Bernard, 2002 Chestnut Street.
1894 Bevan, D., 1015 South Third Street.
1894 Beyea, H. D., 237 South Thirteenth Street.
1892 Birney, D. B., 1810 DeLancey Place.
1893 Bochroch, Max H., 968 North Eighth Street.
1886 Bodamer, George A., 1507 Girard Avenue.
1892 Boger, John A., 2213 North Broad Street.
1895 Bolling, Robert H., Chestnut Hill.
1896 Booth, Arthur W., Episcopal Hospital.
1898 Boston, L. Napoleon, Philadelphia Hospital.
1893 Boyer, H. P., 1338 Spruce Street.
1857 Brinton, John H., 1423 Spruce Street. (O. M.)
1898 Brinton, Ward, 1423 Spruce Street.
1896 Brown, H. MacV., 915 South Forty-ninth Street.
1895 Brubaker, A. P., 105 North Thirty-fourth Street.
1893 Bryan, J. Roberts, 4200 Chestnut Street.
1887 Burr, Charles W., 1327 Spruce Street.

ELECTED

1893 Carpenter, H. B., 1523 Locust Street.
1897 Carpenter, J. T., Jr., 1419 Walnut Street.
1887 Cattell, H. W., 3709 Spruce Street.
1898 Chestnut, J. E., 1757 Frankford Avenue.
1887 Chrystie, Walter, Bryn Mawr, Pennsylvania.
1866 Cleemann, Richard A., 2135 Spruce Street.
1898 Coates, George M., Devon, Pa.
1872 Cohen, J. Solis, 1431 Walnut Street.
1883 Cohen, S. Solis, 219 South Seventeenth Street.
1896 Coley, Thomas Luther, 1339 Pine Street.
1890 Coplin, W. M. L., 1419 South Broad Street.
1898 Currie, Thos. R., 113 East Cumberland Street.
1871 Curtin, Roland G., 22 South Eighteenth Street.

1894 Da Costa, J. Chalmers, 1629 Locust Street.
1896 Da Costa, J. C., Jr., 1633 Arch Street.
1857 Da Costa, J. M., 1700 Walnut Street. (O. M.)
1885 Daland, Judson, 319 South Eighteenth Street.
1857 Darrach, James, 5020 Green Street, Germantown. (O. M.)
1890 Davis, C. N., 1931 Spruce Street.
1882 Davis, G. G., 255 South Sixteenth Street.
1893 Davisson, Alexander H., 2024 Pine Street.
1889 Deaver, H. C., 1534 North Fifteenth Street.
1885 Deaver, J. B., 1634 Walnut Street.
1882 Dercum, F. X., 1719 Walnut Street.
1895 Donnellan, P. S., 1028 Spruce Street.
1894 Dougherty, S. W., 215 South Seventeenth Street.
1888 Downes, Andrew J., 1725 Girard Avenue.
1889 Downs, Norton, 102 West Walnut Lane, Germantown.
1877 Drysdale, T. M., 1305 Locust Street.
1866 Duer, Edward L., 1606 Locust Street.
1876 Dulles, C. W., 4101 Walnut Street.
1883 Dunmire, G. B., 1618 Spruce Street.
1860 Dunton, W. R., High Street and Germantown Avenue, Germantown.

1896 Edsall, David L., 330 South Sixteenth Street.
1891 Eshner, A. A., 224 South Sixteenth Street.

ELECTED

1893 Farr, W. W., 5800 Green Street, Germantown.
1892 Fenn, William B., Denver, Col.
1881 Fenton, T. H., 1319 Spruce Street.
1857 Fischer, Emil, 729 North Sixth Street. (O. M.)
1876 Fisher, Henry M., 317 South Twelfth Street.
1894 Fox, L. Webster, 1304 Walnut Street.
1886 Freeman, W. J., 1720 Locust Street.
1889 Frese, Carl, German Hospital.
1895 Frazier, Charles Harrison, 133 South Eighteenth Street.
1880 French, M. S., 1437 Spruce Street.
1894 French, Samuel, Plymouth, Pa.
1890 Friebis, George, 1906 Chestnut Street.
1896 Fritz, Clarence H., 3932 Brown Street.
1885 Fussell, M. H., 189 Green Lane, Manayunk.

1895 Garitee, C. J., 1117 Spruce Street.
1897 Gerson, T. Perceval, Philadelphia Hospital.
1870 Getchell, Frank H., 1432 Spruce Street.
1894 Girvin, John H., 3912 Walnut Street.
1898 Gittings, T. Claxton, 4013 Chestnut Street.
1890 Gleason, E. Baldwin, 41 South Nineteenth Street.
1882 Godey, H. E., N. E. cor. Nineteenth and Spruce Streets.
1886 Goodell, W. Constantine, 2041 Locust Street.
1890 Grayson, C. P., 251 South Sixteenth Street.
1867 Grier, Matthew J., 1531 Spruce Street.
1883 Griffith, J. P. C., 123 South Eighteenth Street.
1897 Gross, Wm., 701 North Fortieth Street.
1894 Grove, J. H., 1504 Arch Street.
1874 Guitéras, John, 130 South Thirty-ninth Street.
1890 Gummey, Frank P., 4774 Main Street, Germantown.

1896 Haden, H. C., 346 South Fifteenth Street.
1893 Hamill, S. M., 1822 Spruce Street.
1893 Hand, Alfred, Jr., 211 South Seventeenth Street.
1885 Hare, Hobart A., 222 South Fifteenth Street.
1897 Harris, A. L., Jefferson Medical College Hospital.
1898 Hart, Chas. D., Pennsylvania Hospital.
1890 Hartzell, M. B., 3634 Chestnut Street.

ELECTED

1873 Hearn, W. Joseph, 1120 Walnut Street.
1870 Henry, Frederick P., 1635 Locust Street.
1896 Henry, John N., 1635 Locust Street.
1880 Hewson, Addinell, Jr., 1508 Pine Street.
1894 Hiller, Hiram M., 350 South Sixteenth Street.
1894 Hughes, George M., 241 North Eighteenth Street.
1882 Hughes, W. E., 3726 Baring Street.

1890 Izard, William, Sixth and Penn Streets, Camden, N. J.

1890 Jameson, W. B., S. E. corner Fortieth and Brown Streets.
1890 Jarrett, Henry, Camden, N. J.
1895 Johnston, John A., National Soldiers' Home, Virginia.
1895 Jopson, J. H., 334 South Sixteenth Street.
1898 Judson, Chas. F., 1934 Pine Street.
1885 Jurist, L. J., 918 North Broad Street.

1895 Kelly, A. O. J., 1911 Pine Street.
1890 Ketcham, S. Rush, 1708 Green Street.
1897 King, W. H., 412 South Fifteenth Street.
1888 Kirby, Ellwood, 1202 Spruce Street.
1894 Kirkbride, Thomas S., Jr., 1406 Spruce Street.
1897 Klein, Alexander, 513 Pine Street.
1890 Kneass, Samuel S., University Dormitories.
1895 Krusen, Wilmer, 158 North Twentieth Street.
1892 Kyle, D. Braden, 1517 Walnut Street.

1893 Lainé, D. T., 129 South Fifteenth Street.
1890 Laplace, Ernest, 1828 South Rittenhouse Square.
1898 Latta, Samuel W., 3626 Baring Street.
1894 Leach, W. W., 2118 Spruce Street.
1869 Leaman, Henry, 828 North Broad Street.
1888 Leidy, Joseph, 1319 Locust Street.
1887 Leopold, Isaac, 1520 Franklin Street.
1868 Lewis, Francis W., 2016 Spruce Street.
1875 Lewis, Morris J., 1316 Locust Street.
1894 Lincoln, Clarence W., Wayne Avenue and Coulter Street, Germantown.

ELECTED

1893 Lindsay, John, 340 South Fifteenth Street.
1886 Lloyd, James Hendrie, 3910 Walnut Street.
1898 Lodholz, Edward, 3103 Diamond Street.
1894 Loeb, Ludwig, 1337 North Seventh Street.
1886 Longaker, D., 652 North Eighth Street.
1875 Longstreth, Morris, 1416 Spruce Street.

1880 McClellan, George, 1352 Spruce Street.
1892 McFarland, J., 421 West Price Street, Germantown.
1894 McKee, James H., 1519 Poplar Street.
1890 McKelway, George I., 1612 Locust Street.
1894 McLeod, George, 261 South Fifteenth Street.
1898 McReynolds, R. P., 3722 Walnut Street.
1887 Martin, Edward, 415 South Fifteenth Street.
1892 Massey, Frank, 3604 Locust Street.
1864 Mears, J. Ewing, 1429 Walnut Street.
1873 Meigs, Arthur V., 1322 Walnut Street.
1878 Mills, C. K., 1909 Chestnut Street.
1884 Mitchell, J. K., 256 South Fifteenth Street.
1893 Mitcheson, R. S. J., 1522 North Fifteenth Street.
1896 Modell, D. A., 242 Fairmount Avenue.
1857 Morehouse, George R., 2033 Walnut Street. (O. M.)
1898 Morris, Henry, 315 South Sixteenth Street.
1885 Morrison, William H., Holmesburg, Pennsylvania.
1887 Morton, S. W., 113 South Twentieth Street.
1890 Muehleck, G. A., 1320 South Fifth Street.
1869 Müller, August F., 4753 Green Street, Germantown.
1880 Musser, J. H., 1927 Chestnut Street.

1879 Neff, J. S., S. W. corner Twenty-third and Spruce Streets.
1894 Newcomet, W. S., 3229 Powelton Avenue.
1898 Newton, R. D., 2705 Oxford Street.
1861 Norris, William F., 1530 Locust Street.

1894 O'Malley, James, 1104 Jackson Street.
1897 Ostheimer, Maurice, 1229 Locust Street.

ELECTED

1885 Packard, F. A., 110 South Eighteenth Street.
1893 Packard, F. R., 324 South Nineteenth Street.
1857 Packard, John H., Hotel Stenton. (O. M.)
1892 Pearce, F. S., 1407 Locust Street.
1896 Pease, H. D., 5812 Morris Street, Germantown.
1890 Pennock, W. J., 1407 North Seventeenth Street.
1891 Penrose, Charles B., 1331 Spruce Street.
1865 Pepper, William, 1811 Spruce Street.
1897 Pepper, William, Jr., 1811 Spruce Street.
1895 Perkins, F. M., 1428 Pine Street.
1897 Peters, L. C., 2136 Oxford Street.
1885 Piersol, G. A., 4722 Chester Avenue.
1894 Pitfield, Robert L., 4788 Main Street, Germantown.
1893 Posey, W. C., 1831 Chestnut Street.
1890 Potts, C. S., 1629 Locust Street.
1889 Price, Joseph, 241 North Eighteenth Street.

1885 Randall, B. A., 1806 Chestnut Street.
1898 Ravenel, M. P., University of Pennsylvania, Veterinary Department.
1894 Reckefus, C. H., 506 North Sixth Street.
1885 Reed, Boardman, 1831 Chestnut Street.
1894 Rhein, J. H. W., 1330 Spruce Street.
1897 Rhoads, Thomas Leidy, 1703 Walnut Street.
1894 Riesman, David, 326 South Sixteenth Street.
1891 Ring, G. O., 1442 North Thirteenth Street.
1876 Roberts, John B., 1627 Walnut Street.
1884 Robertson, W. E., 912 North Fourth Street.
1889 Robinson, William Duffield, 2012 Mount Vernon Street.
1891 Rosenthal, E., 517 Pine Street.
1894 Ross, George, 637 North Sixteenth Street.

1896 Sailer, Joseph, 330 South Sixteenth Street.
1867 Santee, Eugene J., 605 North Eleventh Street.
1890 Sartain, Paul J., 212 West Logan Square.
1895 Schamberg, J. F., 831 North Broad Street.
1897 Schreiner, E. R., N. E. corner Thirty-eighth and Spring Garden Streets.

ELECTED

1882 de Schweinitz, G. E., 1401 Locust Street.
1890 Scott, J. A., 121 South Eighteenth Street.
1898 Sharpless, Wm. T., West Chester, Pa.
1885 Shober, J. B., 112 South Seventeenth Street.
1889 Shoemaker, Harvey, 109 South Twentieth Street.
1868 Sinkler, Wharton, 1606 Walnut Street.
1881 Skillern, P. G., 241 South Thirteenth Street.
1881 Skillern, S. R., 3509 Baring Street.
1894 Smith, Lawrence S., 133 South Eighteenth Street.
1898 Smyth, Henry Field, Germantown Hospital.
1894 Spellissy, Joseph, 108 South Eighteenth Street.
1896 Spiller, William G., 4409 Pine Street.
1890 Stahl, B. F., 1502 Arch Street.
1894 Steele, J. D., 4504 Chester Avenue.
1889 Stengel, Alfred, 332 South Seventeenth Street.
1889 Stevens, Arthur A., 320 South Sixteenth Street.
1886 Stewart, David D., 108 South Seventeenth Street.
1896 Stewart, Alonzo H., 250 North Twelfth Street.
1857 Stillé, Alfred, 3900 Spruce Street. (O. M.)
1884 Stritmatter, I. P., 999 North Sixth Street.
1869 Stryker, Samuel S., 3821 Walnut Street.
1894 Swan, John M., 807 North Forty-first Street.

1895 Taylor, A. E., 334 South Sixteenth Street.
1896 Taylor, J. Gurney, Pennsylvania Hospital.
1897 Teller, Wm. H., 1934 Green Street.
1894 Thomas, W. Hersey, 1445 North Seventeenth Street.
1894 Thomson, Archibald G., 1426 Walnut Street.
1868 Thomson, William, 1426 Walnut Street.
1897 Tucker, Henry, 19 South Twenty-first Street.
1892 Tunis, J. P., 129 South Eighteenth Street.
1898 Twaddell, Thomas, Cartwright, Elk Co., Pa.
1863 Tyson, James, 1506 Spruce Street.
1890 Tyson, T. Mellor, 1506 Spruce Street.

1895 Vanderwoort, C. A., 3250 North Fifteenth Street.
1887 Vansant, Eugene L., 1929 Chestnut Street.

ELECTED	
1898	Wadsworth, W. S., Presbyterian Hospital.
1891	Wallace, James, 350 South Sixteenth Street.
1890	Warder, Charles B., 1401 North Sixteenth Street.
1874	Warder, W. H., 1212 North Broad Street.
1895	Wendell, W. G., 4126 Chester Avenue.
1887	Westcott, T. S., 108 North Nineteenth Street.
1885	Wharton, H. R., 1725 Spruce Street.
1897	White, Courtland Y., 1808 Diamond Street.
1893	White, F., Elwyn, Pennsylvania.
1873	White, J. William, 1810 South Rittenhouse Square.
1893	Whiting, A. D., 1523 Spruce Street.
1868	Willard, De Forest, 1601 Walnut Street.
1897	Williams, Henry L., 112 South Eighteenth Street.
1898	Willson, R. N., Pennsylvania Hospital.
1869	Wilson, James C., 1437 Walnut Street.
1891	Wilson, Samuel M., 1517 Arch Street.
1891	Wilson, W. R., 112 South Twentieth Street.
1895	Witmer, A. F., 322 South Fifteenth Street.
1890	Wolff, L., 333 South Twelfth Street.
1890	Wood, A. C., 1501 Walnut Street.
1898	Wood, George B., 1925 Chestnut Street.
1865	Woods, D. Flavel, 1501 Spruce Street.
1897	Woodward, E. B., 4222 Chestnut Street.
1897	Yarrow, Thomas J., Jr., Episcopal Hospital.
1892	Ziegler, Jacob L., Mt. Joy, Lancaster County, Pa.
1895	Zimmerman, Mason W., 1633 Chestnut Street.

NON-RESIDENT MEMBERS.

Ashhurst, W. W., Chihuahua, Mexico.
Baines, A. S., Jr., 4484 Finney Avenue, St. Louis, Mo.
Ball, M. V., Warren, Pa.
Breinig, P. B., Bethlehem, Pa.
Brooke, B., Radnor, Pa.
Carter, W. S., Galveston, Texas.
Cerna, D., Galveston, Texas.
Dock, George, Ann Arbor, Mich.
Edwards, W. A., San Diego, California.
Egbert, Joseph P., Wayne, Delaware County, Pa.
Eskridge, J. T., Denver, Col.
Fox, J. M., Torresdale, Pa.
Gaylord, H. R., Buffalo, N. Y.
Hamaker, W. D., Meadville, Pa.
Hamann, C. A., 517 Euclid Avenue, Cleveland, Ohio.
Harrison, W. H., Harrisburg, Pa.
Hatch, John L., 2010 Fifth Avenue, New York City.
Hileman, J. B., Altoona, Pa.
Hunt, J. R.
Jamar, John H., Elkton, Md.
Lincoln, W. R., Cleveland, O.
McCarthy, D. J.
Muhlenberg, Frank, Reading, Pa.
Nancrede, C. B., Ann Arbor, Mich.
Osler, William, Johns Hopkins Hospital, Baltimore, Md.
Pfouts, G. B., Morris Plains, N. J.
Powell, W. M., Atlantic City, N. J.
Rahter, C. A., Harrisburg, Pa.
Shumway, E. A.
Slifer, Henry F., North Wales, Pa.
Smith, Allen J., Galveston, Texas.
Stiles, G. A., Conshohocken, Pa.
Taylor, L. H., Wilkes-Barre, Pa.
Toulmin, H., Milwaukee, Wiss.
Wells, G. M., Wayne, Delaware County, Pa.
Wetherill, R. B., Lafayette, Ind.
Wilmarth, A. W., State Home for the Feeble-minded, Chippewa Falls, Wisconsin.

CORRESPONDING MEMBERS.

ELECTED	
1866	Bumstead, Freeman J. Died 1879.
1859	Clark, Alonzo, Professor of Theory and Practice of Medicine in the College of Physicians and Surgeons, New York. Dead.
1898	Cornil, V., Faculté de Médecine, Paris, France.
1859	Dalton, J. C., Professor of Physiology in the College of Physicians and Surgeons, New York. Dead.
1885	Dent, Clinton T., Assistant Surgeon and Lecturer on Practical Surgery at St. George's Hospital, Surgeon to Belgrave Hospital for Children, London, England.
1860	Ellis, Calvin. Died 1883.
1888	Fedeli, Gregorio, Rome, Italy.
1858	Flint, Austin, Sr. Died 1886.
1890	Gibbes, Heneage, Ann Arbor, Michigan.
1858	Hammond, William A., Member of the National Academy of Sciences, New York.
1860	Isaacs, Charles E., Brooklyn. Died June 16, 1860.
1878	Jackson, J. B. S., Boston. Died 1879.
1886	Pye-Smith, P. H., Physician and Lecturer on Medicine at Guy's Hospital, London, England.
1860	Reeves, James E., Wheeling, West Virginia. Dead.
1861	Rokitansky, Carl. Died 1878.
1860	Watson, John, New York. Died June 3, 1869.
1898	Welch, Wm. H., Professor of Pathology, Johns Hopkins University, Baltimore.
1859	Worthington, Wilmer, West Chester, Pennsylvania. Died 1873.

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Proceedings
of the
Pathological Society of Philadelphia.

OCTOBER 28, 1897. NEW SERIES, VOL. 1, NO. 1.

Address of the Retiring President,

DR. J. H. MUSSER.

Triple feelings of somewhat diverse character struggle for expression on this occasion. They are feelings of regret, of gratification and of gratitude. The first is purely personal. Of the many shortcomings of his incumbency in this office, none create greater feelings of regret, and indeed of mortification, in the president, than the failure to secure for the society the immediate publication of the proceedings in the current issues of the weekly journals. We may point with satisfaction and pride at our transactions as a whole, but the immediate proceedings, with the discussions, should be issued at once. Your president called attention to this dereliction of our duty as a society to the individual members, to the science of medicine and to Philadelphia medicine. It is not necessary to rehearse the ways and means that have been suggested, but I earnestly urge you to uphold the incoming chairman to make this a successful feature of his office-bearing period. Let him not leave the chair with feelings akin to those which I cannot throw off to-night.

The feelings of gratification, which it is my pleasure to express, should be held in common by us all. It should be a source of great gratification that the course of the Philadelphia Pathological Society remains one of great success and usefulness. We should be gratified that the meetings throughout the year have been most largely attended, and that the contributions have been abundant and of high scientific value. Not only is this a source of pride for our society, but for Philadelphia medicine. It is interesting and remarkable, and bodes better for medicine in this city that the interest in pathology is so well sustained. It is so much the better, too that this interest so largely centres in the young men of the profession. It is not necessary to rehearse the importance of the study of morbid anatomy and of pathology. It has been repeated from this chair thrice, and more than thrice, that there is no surer way to solid, substantial success and fame than by the pursuit of these studies.

We should be gratified that our labors here are on the same plane as those in similar societies in this country. You remember there are but three or four societies devoted to pathological work alone in this country. One of my friends in Boston, himself the leader of pathology in that city, and indeed one of the leaders of the country, could not but marvel at the size of the membership of our society, as well as the character of the proceedings. Well may we congratulate ourselves that the society still upholds the promise of its eminent founders, and that its members, past and present, may look upon it with pride as one of the distinctive features of Philadelphia medicine.

Finally, expressions of gratitude arise. These, too, are personal. Gratitude for the loyalty, the courtesy and the uniform kindness you have bestowed upon me during the past four years. No society can fail to be successful, the individual members of which bestow such cordial support and faithfulness upon its chairman. To express my gratitude and my reciprocal feelings under these circumstances would, in cold type, appear fulsome and an affectation. Believe in my sincerity when I say that the intercourse that has taken place during my incumbency in this office, between myself and the individual members has created in me, for each of them an affectionate regard which words fail me to express. May the success of the Pathological Society continue, and may you accord to its new president the same loyalty and devotion it has been the great honor of your retiring president to receive, is my most sincere wish. *October 14, 1897.*

Tumor of the Spinal Meninges.

BY DRS. S. M. MORTON AND A. F. WITMER.

The case is from the wards of the Philadelphia Home for Incurables, where the patient was under observation for more than one year. Age thirty-one: a native of Ireland, admitted October

31, 1895, with the following history: family history negative; good previous personal history. Present illness dates from October, 1893, when she bruised the left breast, following which she complained of soreness and pain in the injured part. Six months later a small, hard mass was felt beneath the skin over the left breast, while pain and soreness at this point were much aggravated.

In November, 1894, the breast was operated upon, and again in February, 1895. Relief from pain was obtained for six weeks after the second operation, when the cicatrix again underwent neoplasmic change, the pain returned, the axillary glands became enlarged, the left arm painful and edematous. At the time of the last operation no secondary formation of new growth had occurred.

On admission to the Home in October, 1895, the patient was very feeble and extremely anemic; the cicatrix remaining after the removal of the breast was the site of hard masses, had begun to ulcerate, and the glands of the left axilla were involved. Small whitish tumors were scattered over the skin about the cicatrix and over the skin covering the right breast; these were not tender or painful.

November 10 a slight projection of the twelfth thoracic vertebra was noticed, at which point pressure was exquisitively painful. While in the sitting posture the patient complained of pain radiating down the right leg. Walking at first possible when fully supported, became impossible early in February, 1896.

The pain also increased in severity; the slightest touch of the right leg causing great suffering. Constipation was always a marked symptom.

The bladder function was undisturbed until two months before death, when retention of urine was first noticed. About six months after admission the right humerus became infiltrated with a new growth; three months later the left tibia was invaded. Shortly before death a bed-sore developed on the sacrum. The patient died from exhaustion on December 17, 1896.

Autopsy held twelve hours after death. Rigor-mortis not marked; left arm and right leg edematous; skin shows scattered masses of dense tissue, varying in size from a pin-head to a walnut; at mammæ and in left axilla ulceration is extensive; sacral decubitus. The right pleura was adherent, the left lung contained a cavity partly filled with purulent matter. No valvular lesion of the heart. There were three or four small calculi in the gall-bladder. The kidneys show suppurative changes, particularly marked in the pelvis; the right also contained two calculi. The suprarenals showed an increased amount of connective tissue. The cavity of the uterus was considerably narrowed, the bladder was suppurative. At the middle third of left leg and in the upper third of right arm the infiltrating growth has involved the bone. The skin over the scalp was normal, the calvarium thickened, the dura non-adherent, but of denser structure than normally. The brain was normal in appearance, the sub-arachnoid space contained an excess of fluid. The membranes of the cord were non-adherent. The twelfth thoracic vertebra was carious, and a soft growth was adherent to the dura at this point.

The growth was of irregularly rounded outline with the long axis situated transversely to the spinal column, and but loosely attached to the dura and spinal fat. This growth measured 33 m.m. in diameter; 25 m. m. in length; 19 m.m. in width, and is elevated about 6 m. m., from the surface of the cord. The cord substance was considerably compressed at this point.

The cord was hardened in formalin and sections stained by the Van Gieson and eosin-hæmotoxylin methods. Microscopical examination of the neoplasm shows the appearances of an endothelioma. The blood-vessels of the cord were generally thickened: the intrima slightly hypertrophied and the media greatly enlarged. The cord substance was intact; the tumor being entirely extra-medullary.

As new growths of the cord and meninges afford no distinguishing symptoms, they are usually classed together. Of 133 cases recently collected the growth was extra-medullary in 97; in 36 of the total number the growth was some form of sarcoma. In size the neoplasm may vary from a pea to a maximum diameter of 75 m. m. The case that we record is of medium size and single.

The symptoms of new growths in the cord are pain, spasm, muscular rigidity, paralysis and anæsthesia. In our case we found marked pain, particularly of the root-variety, paralysis and trophic disturbance.

Bailey, of New York, has recently called attention to tumors of the spinal cord unaccompanied by pain. In one of his two cases the growth was a psamoma, in another a gumma, while a third case reported by Clarke was endothelial.

In our case the growth was also endothelial, but pain was an early and marked symptom.

In the present uncertainty of our knowledge regarding special nerve-tracts for pain, we must conclude with Bailey that the pain is induced by pressure generally distributed, and not to pressure upon any particular part of the cord substance.

We desire to acknowledge our indebtedness to Dr. Shively, of the Philadelphia Polyclinic Hospital, for the preparation of the sections.

September 9, 1897.

References: Starr—American Journal Med. Sc., 1895; Bailey—Journal Nervous and Ment. Dis., 1896; Primel—Nervous Diseases by American Authors, edited by Dercum; Clarke—Brain, 1895.

Discussion.

Dr. Joseph Sailer inquired more minutely into the macroscopic appearances, and discussed the possibility of the neoplasm being an epithelial growth.

Dr. Witmer responded that the relations between stroma and cell-nests commonly seen in cancer were here not present; it was in some cases confessedly difficult to differentiate epithelial from endothelial cells.

A Case of Uremia.

BY DR. AUGUSTUS A. ESHNER.

H. G., an unmarried female servant, 52 years old, was admitted to the Polyclinic Hospital on September 4 in a state of coma. The only history that could be gained was that she had been unconscious since the day before and that she had had an attack similar to the present one some five months previously. The pulse was somewhat accelerated (124 in the minute, reaching, however, on subsequent observations as high as 134, and not falling below 120). The respirations on admission were 16 in the minute, although they subsequently rose as high as 30. The temperature was 101.4°, but it soon rose to 104.2°, in the neighborhood of which it hovered during the twelve hours preceding death. The heart-sounds were somewhat ill defined, but no murmur could be heard. The pulmonary percussion-resonance was impaired upon both sides and moist rales could be heard in places. The pupils were approximately normal in size, the left a little the larger. There was general muscular relaxation, but no evidence of actual paralysis, local or general. The breathing was quiet, the cheeks flapping passively with inspiration and expiration. There was a free, purulent discharge from the vagina. At times twitching of the muscles of the right side of the face was noticed, and at times also, of the right hand and arm. On examination of the urine, which was greenish-yellow in color, slightly alkaline in reaction, with a specific gravity of 1016, reactions for sugar and albumin could not be elicited. Microscopic examination, however, disclosed the presence of some granular tube-casts and vaginal epithelium. Some doubt existing at first as to the nature of the case and with the possibility of some form of narcotic poisoning especially in view, strychnin sulphate, gr. 1-10, was administered hypodermically, and the stomach was freely washed out by the resident physician, soon after the patient's admission into the hospital. Later in the day, as the nature of the case became more clear, the patient was put in the hot-air bath, but did not perspire freely. Six drops of croton-oil were given in divided doses, but failed to induce catharsis. In addition, tincture of digitalis and spartein sulphate were injected hypodermically, and as a final resort two pints of normal salt-solution were introduced into the subcutaneous tissues of each mammary region. The patient failed to return to consciousness, and died some nineteen hours after her reception into the hospital. The post-mortem examination was made by the coroner's physician, Dr. Morton, through whose courtesy I had the privilege of seeing the specimens.

The lungs were adherent, the left to a much more pronounced degree than the right. Both presented posteriorly and inferiorly considerable areas of consolidation, dependent in part upon congestion and edema, and in part upon broncho-pneumonia. They also exhibited a moderate degree of pigmentation and were emphysematous at their margins. The heart was normal in size, and its cavities and muscular wall exhibited no gross abnormality. It was rather freely supplied with fat. The leaflets of the several valves exhibited inconsiderable thickening, obviously insufficient to occasion appreciable functional derangement. In the cavities and in the large vessels was found a moderate amount of fibrinous clot, partially decolorized. The kidneys were, perhaps, a little increased in size; their capsules were adherent, and on removal exposed a granular surface. The normal relation between the cortex and the medullary structure appeared maintained. The cut section was pale and exhibited linear streaks of fatty degeneration. The liver was pale and exhibited no other peculiarity. The spleen was small, its capsule thickened and its parenchyma marked by distinct fibrous trabeculæ. The uterus was retroflexed and the seat of several small fibroid tumors as well as of a catarrhal inflammation of its mucosa. The oviducts were normal; the ovaries sclerotic. The brain was intensely injected, but free from gross lesion.

The case is interesting, first, on account of the doubt surrounding the diagnosis. The patient was brought into the hospital comatose and under conditions that required consideration of the possibility of some form of narcotic poisoning. It is true no characteristic odor could be detected about the patient and the pupils were not contracted, but both of these signs might be wanting in an actual case of opium poisoning. There might be no odor at all present, or this might be masked by something taken at the same time as the poison, or subsequently, or possibly even previously. Then the patient might come under observation before the effect upon the pupils had become manifest.

The differentiation between uremia and cerebral hemorrhage is at times one of the most difficult problems in clinical medicine. The primary shock attending an extravasation of blood into the brain may occasion general muscular relaxation and localizing phenomena may be wanting, while as a matter of fact the latter are by no means rare in cases of uremia. So, too, the presence of albumin and tube-casts in the urine is not conclusive. The conditions upon which these phenomena depend are likewise often just those that predispose to the occurrence of cerebral hemorrhage.

Finally, a word may be said concerning the presence of fever in the case here reported. It has been taught that the uremic state is usually attended with a normal or subnormal temperature, but clinical experience shows that in a very considerable proportion of cases the temperature becomes distinctly febrile. As has been well pointed out recently by Stengel,* fever may occur in the course of nephritis: at the onset of an acute attack; as a result of the infection or intoxication causing the disease or of the inflammatory or degenerative lesions of the kidneys themselves; in the course of acute or chronic nephritis, as the result of various complications; and in the uremic state. The fever associated with uremia may be pronounced and sudden in onset and attended, with coma, delirium and convulsions; or it may be more gradual, more lasting, less marked and associated with typhoidal symptoms. In the case here reported the inquiry suggests itself as to whether the broncho-pneumonia present was an untoward result of the use of the stomach-tube. The pulmonary lesion was evidently of longer standing than to be thus accounted for. It is to be borne in mind, further, that the pneumonia was probably not without some influence in bringing about the elevation of temperature. A rather noteworthy feature, already referred to, was the comparative infrequency of breathing.

September 9, 1897.

DISCUSSION.

DR. F. A. PACKARD pointed out the prevalency in text-books of the erroneous teaching that a subnormal temperature is the rule in uremia, and emphasized the frequency with which high fever occurs.

DR. J. P. ARNOLD.—I wish to call attention to the occurrence of local tissue-necrosis as a result of hypodermoclysis. I saw in the Presbyterian Hospital a case in which most marked sloughing had been produced by the procedure, due, I believe, to the injection of an excess. Since then I have read other reported similar cases. Small multiple injections should be employed.

DR. ESHNER agreed with Dr. Packard regarding the matter of fever in uremia. He desired to call especial attention to the relative low rate of respiration in this case, contrasted with the heart beats.

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*American Journal of Medical Sciences, November, 1895, p. 92.

Primary Cancer of the Gall-bladder—Extensive Secondary Involvement.

PRELIMINARY COMMUNICATION.

BY DR. DAVID RIESMAN.

The patient was a married woman, thirty-eight years of age. About November, or December, 1896, after an attack of rheumatism, she was seized with vomiting and epigastric pain, which, with intermissions, continued until I saw her, in March, 1897.

She then was still fairly-well nourished, suffered from vomiting, constipation, and intense thirst, and had a slight jaundice. The abdomen was distended and tender, the liver was enlarged, and nodules could be felt on its surface and along the edge. There was no enlargement of the spleen. Signs of effusion in the peritoneal cavity were observed. The knee-jerks were exaggerated, and slight ankle-clonus was present. The temperature varied from 101° to 104° F. A blood-count showed 4633330 red corpuscles and 31250 leukocytes, a ratio of white to red of 1:148.

There was marked edema and ascites; for the relief of the latter tapping was resorted to and 5½ pints of fluid were withdrawn. An examination of the fluid revealed the presence of bile, a trace of sugar, and albumin to the amount of 2.67 grammes in 100 c. c. The specific gravity was 1013. Microscopically, nothing save red corpuscles were seen. Culture-tubes inoculated with fluid remained sterile. A blood examination made just before the tapping showed red corpuscles, 4,500,000; white corpuscles, 55,125 (ratio of white to red of 1:81.6); hemoglobin, 60 per cent. In stained preparations the increase in leukocytes was seen to affect principally the multinuclear variety.

The patient sank gradually and died, after a few days of delirium, of exhaustion. At the autopsy the *liver* was found much enlarged and studded throughout with cancer-nodules. A number of secondary growths were seen on the peritoneum, including that covering the diaphragm. The *gall-bladder* was small, contracted, and adherent to the liver. Its cavity was greatly reduced in size —about large enough for a walnut—and contained a single dark-colored gall-stone. The wall was enormously thickened—at its thickest part measuring about ¾ inches—and soft, friable, shreddy and disorganized. The cancerous tissue surrounded the cystic duct, but did not occlude it. The *pancreas* contained a cancer nodule the size of a large cherry at the point of junction of the head and neck. The lymphatic glands along the lesser curvature of the stomach were cancerous. The *spleen* was slightly enlarged, but free from metastatic growths. The *lungs* were studded with cancerous growth, and a large bundle of cancerous lymph-glands surrounded the great vessels arising from the heart.

Microscopy.—The tumor of gall-bladder is a cylindric epithelioma, with abundant development of the fibrous stroma. The liver tissue is greatly congested, and shows areas of round cell infiltration. As far as the secondary growths in it are concerned, they well represent the features of the cylindric carcinoma. The pulmonary nodules are like those of the liver; there is also an increase in fibrous tissue in the lung.

Remarks.—Of considerable interest, disregarding for the moment the relationship of the gall-stone to the cancer, is the blood-count, which showed a high degree of leukocytosis and oligochromemia. A few words may be devoted to the results of the analysis of the ascitic fluid. Are we able, from such an analysis, *intra vitam*, to determine the cause of the ascites? This is a question about which much difference of opinion exists. Ott (1) in his studies came to the conclusion that no disease had a characteristic amount of albumin. In his cases of cancer of the peritoneum, the amounts varied from 1.799 to 5.203 per cent. The specific gravity was likewise found by him of no aid in diagnosis, and bore, furthermore, no constant relation to the amount of albumin. Runeberg (2) takes a somewhat different view, dividing the effusions into (i) those caused by affections of the serous membrane itself; (ii) those produced by venous stasis; and (iii) those dependent on an excessively hydremic state of the blood, he finds that the albumin-content in these three groups differs rather widely. In group (i) it varies from 4 to 6 per cent; in group (ii) from 1 to 3 per cent; in the third it rarely reaches 0.5 per cent. Combinations of two or all conditions lead to variations in the amount of albumin.

Another feature of interest is the cancerous disease of the glands in the superior mediastinum. It is not unusual to see this involvement in malignant disease originating below the diaphragm.

Light is thrown on the relationship between these glands and the abdomen by some observations of Herbert Durham (3), who noticed enlargement of these glands in animals after artificial intraperitoneal inoculation. The lungs might, of course, act as intermediaries in the transportation of the infectious or neoplastic material.

The last point to which I wish to allude is the relation of the gall-stone to the cancer. Musser (4) in his masterly thesis, has thoroughly dealt with this question. He inclines to the view, which is shared by most writers, that gall-stones are a causal factor, their presence producing a mechanical irritation of the wall of the gall-bladder. This view gains support principally from two facts—first, the great frequency with which stones are found in conjunction with primary cancer of the gall-bladder. In Musser's 100 cases, 69 had gall-stones; Siegert (5) collected 101 cases with gall-stones in 95 per cent.; Kelynack (6) found stones in all of his four cases; and Collinet (7) reports two with lithiasis in each. Secondly, the rarity of stones in cases of secondary cancer—Siegert, in 13 cases (11 of them his own) found them in but 2, or in between 15 and 16 per cent. *September 23, 1897.*

REFERENCES: (1) Ott.—Fritsdrift F. Heilkunde xvii, Heft 4, p. 283. (2) Runeberg.—Berl. Klin. Woch., August 16, 1897. (3) Durham.—Jour. of Pathol. and Bacteriol., March, 1897. (4) Musser.—Trans. of Association of American Physicians, 1889, Vol. iv. (5) Siegert.—Virchow's Archiv, 1893, 132, p. 353. (6) Kelynack.—Practitioner, 1896, 96, p. 387. (7) Collinet.—Bull. Soc. Anal. de Paris, 1892, Juillet, p. 476.

DISCUSSION.

DR. A. E. TAYLOR.—The diagnostic value of quantitative estimations of albumin in ascetic fluids is complicated by the fact that different men employ different methods. Some precipitate by heat, others with ammonium sulphate, or picric acid or tannic acid, others calculate the albumin from the nitrogen present; these methods are not all of equal value, and in different cases the results they furnish will vary markedly, as I have learned from personal experience. But even with one method employed by all, while the results of different men could then be compared, I do not think many positive diagnoses could be made upon the basis of the quantity of albumin in ascetic fluids.

DR. J. H. MUSSER.—The history of the literature of cancer of the gall-bladder is very interesting. Prior to 1880 there were two papers on the subject, since my paper many treatises have appeared. Either these growths were in former years overlooked or else they have become more frequent. Perhaps a greater frequency may have been the result of the tendency of people to lead more sedentary lives. The question is very interesting in its relation to gall-stones, which are much more frequent in these cases than in cases of cancer in other parts of the body. It seems to me probable that gall-stones can produce a cancerous proliferation of the epithelium of the gall-bladder. That the altered physical and chemical conditions in cases of cancer of the gall-bladder might favor the formation of calculi, is also probable. Most cases are in women, 65 per cent. Like this case, my cases had fever, of an intermittent type.

Dr. Riesman.—Metastasis was marked in this case, but the routes are difficult to fix. I believe, too, that there are no constant relations between the causes of ascites and the albumin in the fluid.

Coccidium Oviforme.

BY DR. J. H. McFARLAND.

A few weeks ago my attention was directed toward this peculiar parasitic organism by the outbreak of a fatal disease among the rabbits kept at the laboratory for experimental purposes. The animals had been for some time kept in a certain pen, and had remained well and seemed comfortable. It was after the addition of a large number of new rabbits from a dealer in the city that our pen became affected and the disease manifested itself.

The rabbits suddenly became ill, with diarrhea vomiting, and emaciated, became very weak and ultimately fell into a semi-comatose condition, with frequent convulsive movements, and died after two or three days' obvious illness.

Autopsies upon these animals showed the most marked lesions to be seated in the liver, which was enlarged, hyperemic, dark-colored and soft, and presented a large number of pale elevated new growths, varying in size from a pin-head to a pea, sometimes single, sometimes aggregated in groups, and much resembling in general aspect the lesions of tuberculosis.

In addition to the changes in the liver, the intestine was hyperemic, mottled, thickened here and there, and filled with semisolid contents. There were no formed feces. The peritoneal surface was somewhat injected. Other than these there were no macroscopic changes.

When the liver was incised the lesions ceased to resemble tubercles. Close examination showed that the knife did not pass through a solid tissue like a tubercle, but that it had divided a smaller or larger group of contiguous cysts filled with puriform, rather creamy content that readily escaped. When the empty cavities were carefully scrutinized the structure became somewhat complicated. The cavities occupied a rather dense, pale tissue, were very small as a rule, and were separated from one another by thin, villous, sometimes smooth walls. The cavities seemed to communicate with one another. In general the structure resembled that of some of the epithelial tumors—adenoma and cyst-adenoma.

When the contents of the cysts were subjected to a microscopic examination, the diagnosis at once became clear, for in the puriform matter the adult forms of coccidium oviforme were abundant and readily recognized.

The parasites have been known for more than half a century and as early as 1854 Remak pointed out a resemblance between them and the Psorosperms just discovered by Müller.

The coccidium oviforme is an ellipsoid or egg-shaped unicellular animal measuring about 15–25 ; 30–40μ. They are surrounded, in the adult form, by a very delicate outer and a very resistant, double-contoured, highly-refracting inner membrane.

The contents of the membrane vary in appearance according to the stage of development of the parasite. The adult form consists of a granular mass that entirely fills the capsule or forms a rounded mass in the center of the capsule leaving the poles empty. There is a small micropyle at one end. The appearance of the adult is strikingly similar to the ova of some of the entozoa, especially the distoma lanceolatum, and probably because of this resemblance, in spite of the extended knowledge, of the life history of the parasite, we now possess, there are some who cannot give up the idea that the coccidium is only a stage in the development of one of the intestinal worms.

In the cyst contents from the liver, and in the intestine, large numbers of the parasites in the adult stage are found. When discharged from the body in this form a varying length of time elapses before further development takes place. If the organisms are watched in the "hanging-drops" in the course of one or two weeks a characteristic cycle of development can be observed. The protoplasmic mass constituting the body substance of the organism divide into two, then into four segments which are at first round but which later become elliptical. The segments are provided with their own membranous investiture. In each of the segments, which may be called a "spore," there ultimately develop two falciform embryos of peculiar knobbed shape. A remnant of the original granular protoplasm remains connected with them.

The majority of the organisms that are discharged with the feces are of the adult form and are in a stage that might properly be called potential or latent.

I have not been able to determine that the cycle of development thus described takes place in any of the organisms in the body of the animal. There must, however, be some way in which the coccidia that gain entrance into the body can progressively infect it. There must, therefore, be some other means of multiplication, for this progressive and rapid infection cannot be by cell division as the sperozoa do not divide.

R. Pfeiffer observed that certain of the intracellular parasites instead of being transformed into the perfect or adult coccidia, grow into large rounded protoplasmic masses, very granular 12–15μ in diameter, surrounded by a thin shell or membrane. The nucleus of the mass is obscured by the highly granular nature of the protoplasm but can be discovered by the use of reagents. The transformation of this body into spores is independent of the attainment of any particular size. The sporulation is ushered in by changes in the nucleus suggestive of the wreath figures of karyokinesis. Numerous segments are the result of the nuclear division and sometimes are arranged in geometrical figures within the cell. These nuclear segments elongate and are directly transformed into the falciform embryos. The embryos thus found are delicate ameboid bodies devoid of any distinct power of resistance. They leave the sporocysts and penetrate into the epithelial cells of the bile-ducts where they grow either to adult coccidia or transform themselves into new sporocysts.

This brief description of the processes by which the creatures propagate themselves will make clear the brief account of their life history.

Attempts to produce coccidiosus by allowing animals to feed upon the diseased livers of rabbits dead of the disease, and the ingestion of fecal matter from the infected animals, invariably fail, the organisms being destroyed in the stomach. Animals are, therefore, not directly infected with the discharges from the diseased animals. In order that the infection occur it is necessary that the coccidia containing fecal or other matter be discharged upon the soil of the rabbit warren and for some time kept moist. This allows the potential or latent parasite to undergo the cycle of development first described and form spores containing four sickle-shaped embryos. If at this stage the coccidium be ingested, the outer shell is digested off in the stomach and the embryos liberated from the spores. The embryos seem to have the power—they or their spore cases—of resisting the gastric juice and arriving in healthy condition in the intestine from which the infection certainly starts.

The embryos seem to ascend the ductus choledochus and ductus hepaticus to the smaller branches in which they enter the epithelial cells.

During the entire period of growth the organisms are intracellular parasites of epithelium. They may be single or multiple in the cells, grow at the expense of the cells, and are declared by Leuckart to be members of the karyophagic group of parasites. Their growth within the cells naturally distends them and causes their protoplasm to become a surrounding envelope, which getting more and more attenuated as the coccidium grows, ultimately ruptures and allows the exit of the parasite.

The embryos escaping from the sporocysts are at once able to penetrate and infect the neighboring epithelial cells, and are the cause of most of the trouble. The adult parasites—permanent forms—are carried to the intestine with the bile and discharged with the feces, to undergo the slow cycle of development first pointed out.

In the infected animals two organs show signs of primary disease, the liver and the intestine. The extent of the changes and their exact nature will vary according to the age of the animal. In young rabbits that suffer from the acute fatal form of the disease, the liver is enlarged, hyperemic, softened and occupied by very numerous grayish or whitish nodes resembling tubercles. Externally there are no differences which would at once differentiate them from tubercles.

On section, however, the nodes are found to be of a somewhat cystic nature and to contain a puriform material of whitish color. When evacuated and the tissue scraped clean, it is found that each node is a complex lesion made up of either a number of small cavities or one multilocular cyst with fibrous partitions. The contents of the cyst, which consists of pus, bile, desquamated and disintegrated epithelial cells and coccidia are at once diagnostic and have already been discussed.

When the liver is sectioned and examined microscopically, it is found that the diseased areas at first glance resemble a neoplasm—cystic-adenoma—very closely.

When, however, the lesion is given careful attention, it is seen that the cysts are dilated biliary passages, greatly increased in number and size and surrounded by a recently formed connective tissue rich in cells. In some places the bile-ducts appear comparatively normal in number; in other areas they are normal in appearance and size, but considerably increased in number, as in cases of cirrhosis of the liver. In still other areas, where the disease is most marked, the ducts are widened into true cysts whose epithelium is proliferated and occupied by parasites or destroyed and replaced by masses of them.

Some of the cysts have numerous, long, papillary projections, causing one to think of the papillæ that form in cyst-adenoma of the papilliferous form, or the rougæ of the Fallopian tube. It is a question just what explanation one shall offer for the existence of these. They may be polypoid in nature, like the polypi that develop upon chronically inflamed gastric mucous membranes, or they may be, as Leuckart thinks, simply the remnants of what were once considerable tissue partitions between the bile-ducts, now reduced by the distension to papilla-like out-growths.

Everywhere in the diseased tissue the coccidia in various stages of their development are to be found.

In the more newly affected bile-ducts the original columnar epithelium can be traced without much difficulty. When the disease has lasted longer, cell masses from irritative out-growth of the cells may be found. In the oldest areas all the cells may be lost and masses of adult coccidia are found surrounded by fibro-connective tissue walls.

In old rabbits the infection assumes a more chronic form that is rarely fatal. Few of the sporocysts are formed and the irritation of the bile-ducts produces active connective tissue and new-formation, so that the areas are dense, solid, and cirrhotic, without the cystic dilatations, and characterized by smaller groups of closely approximated parasites, surrounded by quite dense connective tissue masses.

The second organ is the *intestine.*

Being upon a surface instead of in small passages ramifying through a solid organ, the intestinal lesions are readily exposed to view. The infection takes place in the epithelium. Leuckart was able to demonstrate intra-cellular parasites as small as 9-10μ, containing a comparatively large and clear nucleus-like spot, enclosing nucleoli and easily distinguishable from the cell contents, though devoid of a special membrane.

The number of germs finding their way into the epithelium is so considerable that the cells are infected with parasites of all ages and sizes. Often one finds two or more parasites in a single cell. In his studies of the intestinal coccidia of the dog and cat, Leuckart satisfied himself that the parasites remained in the epithelial cells until perfectly ripe. In the milder cases the coccidia are scattered in groups which may be recognized by the naked eye as somewhat elevated spots of varying size. There are, however, other cases in which the surface of the intestine is uniformly infected and considerably swollen, so that it looks as if overlaid with a false membrane.

In the dog, which Leuckart chiefly investigated, the parasites were usually most crowded in the villi, which then appear like little white points, as after an absorption of chyle. Sometimes the Lieberkühnian glands are filled with them and enlarged to double their normal size. The surrounding tissue is inflamed and infiltrated, and sometimes degenerates in small but distinct ulcers. At one time Leuckart thought that in the rabbit the intestinal infection was due to a separate variety, which he named *coccidium perforam.* Now, after so many cultivation, experiments it is known that although the hepatic and intestinal coccidia differ slightly from one another in form, they are identical. The intestinal coccidia measures 13–20: 24–35μ, and according to Kruse is characterized by a less compressed form. Its spores are also said to develop in 3 to 4 days. The chief arguments in favor of their identity are the similar developmental cycle, and their almost invariably simultaneous occurence.

It is to the catarrh of the intestine with its accompanying destruction of epithelium, swelling and inflammation of the submucosa that the symptoms of the disease are chiefly due, and knowing the intestinal lesion, one is not surprised to see that the animals suffer from dyspepsia, anorexia, impaired nutrition, colic, looseness of the bowels, and a final exaggeration of all these sufficient to produce a fatal outcome.

The disease, especially the chronic form, is extremely common among rabbits. The acute coccidiosus is epidemic in its occurrence, and is seen in the young animals in the warren, generally affecting those between the ages of 4 to 6 weeks, just finishing the sucking period and beginning to take green food. In certain badly infected warrens all the rabbits die when this age is reached, or if a few survive, they are slow in developing, perhaps dwarfed in size. In these young animals the falciform embryo and sporocysts are common.

Infected warrens must be abandoned, or a most careful and persistent isolation of the diseased animals practiced, together with disinfection of the earth of the warren, in order to stamp out the disease.

The coccidium oviforme is the common form found in the rabbit. Separate forms occur in the mouse and rat, dog and cat, calf, salamander, and in many of the invertebrate animals, as the myriapodia, cephalopoda, gasteropoda, etc.

Unfortunately, the disease is not strictly confined to the lower animals, but sometimes occurs in man. Thirteen cases are reported in the collection of Kruse in Flugge's new work, "Die Microörganismen." *September 23, 1897.*

Discussion.

Dr. F. A. Packard remarked that he had attempted the preservation of coccidia in glycerine, with the result that the cell-substance was rendered entirely invisible.

Dr. Alfred Hand presented the organs of a child the subject of *acute general miliary tuberculosis.*

Tumor of the Dura Mater.

BY J. P. ARNOLD.

The specimen presented was obtained from a patient in the Presbyterian Hospital. The man had been under observation for the past nine months. His family and personal history were negative. There was no history or signs of syphilis. The onset, two years ago, was marked by severe headache, followed by attacks of unconsciousness of rather short duration, accompanied by loss of power in the right side involving the arm and leg.

When admitted he had almost complete loss of power in the right arm. After grasping the hand the flexors could not be relaxed. Attempts to do so increased their contraction. The headache was constant and increasing in intensity. Both optic disks showed signs of neuritis. There was almost complete loss of vision in the left eye. The paralysis increased until hemiplegia was complete and accompanied by marked interference with speech. The man died of a septic pneumonia.

The tumor may be seen to spring from the dura in the longitudinal fissure of the cerebrum. It is ovoid in shape and its dimensions about 6 x 4 c. m. It displaces the brain tissue outward, and evidently seriously interfered with the functions of the leg, arm and face centers. On section the growth has the appearance of sarcoma. The microscopical characters of the growth will be reported later.

September 23, 1897.

Mitral Stenosis.

BY M. H. FUSSELL.

The patient was seen in June, 1897, with Dr. R. R. Bunting. At that time the patient was suffering from extreme dyspnea, general edema, cyanosis, and rather profuse hemoptysis, which had lasted for one week.

There was a history of severe rheumatism. The following physical signs were noted: Apex beat of heart was in the fifth interspace left of sternum, superficial heart dullness to fourth rib, left of the nipple line; heart's action irregular and rapid; at base no murmur; a rough presystolic murmur with accentuated first sound. Pulmonary second sound markedly accentuated. There was distinct dullness over base of the left lung.

Post-mortem.—Feet and legs were markedly edematous. Marked jaundice of conjunctiva, much subcutaneous fat, all the tissues bile stained. Puncture of first right costal cartilage causes a flow of bloody serum. On turning up the sternum the left pleural cavity was seen to contain a small amount of liquid. The apex of heart was in the sixth interspace. The right pleural cavity contained about two quarts of deeply blood-stained serum. The right lung was collapsed in the apex of the chest cavity. There were a few clots of blood in the right pleural cavity. The lower lobe of right lung was the seat of carnification. The pericardium did not contain an abnormal amount of liquid. The right auricle of heart and the right ventricle were distended with a jelly clot. The wall of the *left* ventricle measures one-half inch.

The heart weighs 1½ pounds. The walls of right auricle are not thickened, but the cavity is very much dilated. The left auricle is filled with a white clot, very firmly organized and adherent to the sides of auricle. It is impossible to insert a finger into the mitral orifice so great is the stenosis. The left lung is badly congested, but crepitant. The right lung is entirely collapsed. The lower lobe is carnified. The spleen was normal in size and not softened. The liver was enlarged and the seat of cardiac cirrhosis. The kidneys were markedly congested. The capsules were not at all adherent, but there were groups of depressions alternating with elevations. The elevations had a markedly granular appearance while the depressions were marked with stellar congested vessels. The narrowing of the mitral orifice is due entirely to the adhesion of the edges, the valves being but little thickened. The right ventricular orifice admits five fingers. The walls of the left auricle are markedly hypertrophied, showing on the inside large bands of muscular fibre, much like the papillary muscles of the ventricle.

September 23, 1897.

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TABLE OF CONTENTS.

SAILER, Heart with Two Ventricular Aneurisms.—**RIESMAN**, Cancer of the Stomach.—**MUSSER**, Melanotic Sarcoma of the Liver.—**SWAN**, Tuberculosis of the Kidney.—**ROBERTS**, Specimen of Internal Strangulation of the Small Bowel by a Thread-like Band.—**MUSSER**, Cerebral Hemorrhage.—**SAILER**, Abscess of Lung.—**CATTELL**, Carbolic Acid Poisoning.—**SWAN**, Dissecting Aneurysm of Aorta.—**SCHREINER**, Gastric Carcinoma; **ARNOLD**, Gastric Carcinoma.

Heart with Two Ventricular Aneurisms.

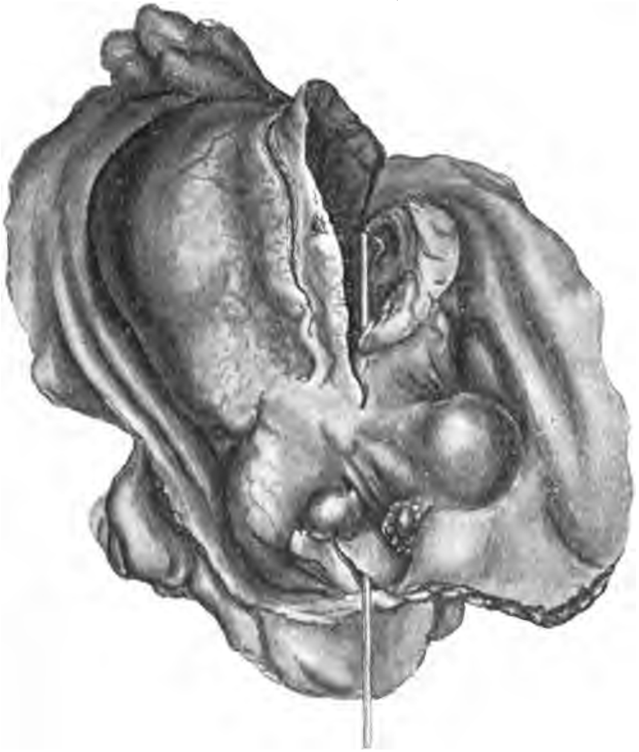
FROM THE PATHOLOGICAL LABORATORY OF ST. JOSEPH'S HOSPITAL.

JOSEPH SAILER, M. D.

This case presented the following pathological conditions: Two aneurisms springing from the left ventricle, one of which had ruptured and caused hemopericardium; chronic interstitial myocarditis, co villosum, cirrhosis of the liver and kidneys, chronic productive gastritis, and congenital hypertrophy of the cecum and appendix.

The clinical history of the patient is as follows: E. C.—white, male, forty years of age; family history negative. He denied syphilis and alcoholism. He had suffered occasionally from attacks of pain in the muscles and joints, that did not keep him from working. About a year ago he began to notice pain and distress in the region of the heart after severe exertion. Three weeks before death he had an attack of severe headache with pain over the precordia, and extreme dizziness; during which he fell and became unconscious Similar attacks occurred frequently and became more severe, and on th night of his admission to the wards of Dr. Henry Morris he had eight. An attack carefully observed by Dr. Smith has been described as follows: There was at first some dizziness, then the muscles were suddenly contracted, the patient assuming the opisthonotic position and remaining rigid for about a minute; this was followed by general relaxation, rapid respiration and extreme bradycardia, which soon changed to rapid running pulse. The skin was covered with a cold sweat. During the tonic convulsion unconsciousness was complete, and afterward the patient was restless and not entirely rational for an hour, and complained of pain over the heart, sometimes extending down the left arm. Physical examination revealed increased area of cardiac dullness, peculiarly irregular at the apex. The apex beat was diffuse, powerful, and felt at its maximum intensity in the sixth interspace one inch outside the nipple-line. The heart's action was slow and irregular, and occasionally there was an imperfect systole. Upon ascultation, a loud, blowing, systolic murmur was heard all over the heart. The urine contained albumin and casts. The epileptiform attacks decreased in frequency under treatment, occurring only every other night while he was in the hospital, but during one on October 27, he suddenly fell back dead.

Autopsy was performed November 29th, 37 hours after death and the following conditions found: There were numerous scars over the anterior surface of the tibia, otherwise no marks of injury. Upon opening the abdominal cavity the cecum was found to be enormously distended; the appendix was about 15 c. m. in length and attached to the mesentery by fibrous adhesions. There was a moderate amount of clear yellow ascitic fluid. Upon opening the thoracic cavity an enormous pericardial sac was found that extended from about 3 c. m. to the right of the sternum to the left mid axiliary line and from the second to the seventh rib. Both lungs were loosely adherent to the thoracic walls and the right also to the pericardium and diaphragm. They were firm, dark, not edematous and the left partially collapsed. Both pleural cavities contained a small amount of liquid, in which were some flocculi of lymph. Upon opening the pericardial cavity it was found to be filled with pure blood in which, particularly near the diaphragmatic surface, were numerous current-jelly clots; the parietal surface was smooth, the visceral surface was thickened and rough, and upon the anterior surface of the left ventricle were some hard dense whitish villi. There was a moderate amount of subepicardial fat and the vessels were slightly tortuous. The apex of the left ventricle showed a sacular elongation about 5 c. m. long and 3 or 4 broad. This was adherent to the diaphragmatic surface of the pericardium and exhibited a small rent at its lower end, from which some reddish globular thrombi protruded. Lying posteriorly to this, but not adherent to the pericardium, was a second sac not quite so large, which seemed to be almost solid. Upon opening the right ventricle the endocardium was found to be normal; the tricuspid orifice admitted four fingers; the pulmonary valve was normal; the mitral orifice admitted two fingers; the aortic valve was normal. Upon the aorta a number of small spots of atheroma were observed. The myocardium of the left ventricle contained several large white fibrous bands. It was red and firm. The two aneurisms, before described, projected from the anterior and posterior



The rod passes through the posterior aneurism and appears in the open left ventricle. The rupture is indicated by the bunch of clots just above. Anterior aneurism to right.

surfaces of the apex of the left ventricle with which they communicated by several openings, each of which admitted a grooved director easily. The orifices of the coronary arteries were not contracted.

On opening the posterior aneurism it was found to consist of two cavities separated by a thick white tissue of cartilaginous consistency. It contained a few soft granular clots. The wall of the posterior section was quite thin; that of the anterior section thick and firm. The communication of the latter with the cavity of the ventricle was by an opening nearly ¼ of an inch in diameter. The interior of the auricles was apparently normal; the foramen ovale was patulous, the slit-like orifice between the two auricles being nearly ½ an inch in length. The intima of the coronary arteries was smooth as far as their caliber permitted of dissection. Neither near the apex nor in any of their branches was there any sign of thrombosis or of endarteritis. The ductus arteriosis was not patulous. The thoracic and abdominal portions of the aorta contained numerous yellowish points of atheroma, but no extensive degeneration. The vessels were of normal elasticity. The lumbar arteries originated from common openings. The bronchial and mediastinal glands were moderately enlarged and very anthracotic. The spleen was of normal size, firm, dark, with hyperplasia of the lymphoid follicles. A small supernumerary spleen was present. The suprarenal capsules showed absence of the central cavity and fatty degeneration of the cortex. The kidneys were of normal size, the color was reddish yellow; the substance was firm; the capsules stripped with some difficulty. The ratio of cortex to medulla was about 1.5. There were numerous cortical cysts containing grayish yellow fluid and a few moderately depressed irregular scars, that rarely extended into the medulary portion. The liver was slightly enlarged, granular, on section "nutmeg," and of dense consistency. On the posterior portion of the convex surface was a much depressed stellate scar. The stomach was remarkably small, and the antrum pylori contracted to about the caliber of the ileum. The mucous membrane was thickened, forming prominent rugae that transversed the organ longitudinally. The mucous membrane of the small intestines appeared to be normal. The brain showed some postmortem softening but no lesions.

The microscopical examination of the myocardium at the level of the largest masses of fibrous tissue shows the characteristic appearance of a chronic interstitial myocarditis. The muscle cells are in part preserved, but many of them exhibit faintly staining nuclei. Others stain poorly and show loss of striation. Between these bundles of muscle cells are thick bands of fibrous tissue with abundant round-celled infiltration. The vessels in the midst of these cicatrices are moderately sclerotic. There is slight thickening of the intima; no extensive proliferation of the adventitia; and the lumen is preserved in all cases, but the number of blood-vessels is less than would be found in the same area of cardiac tissue. The sclerotic changes appear to be diffuse, that is to say, they are not more excessive near the pericardial than near the endocardial surface. The fibrous tissue, however, is not uniformly distributed, being in some places aggregated in masses, in others reduced to delicate strands.

The results of the post-mortem examination explain very satisfactorily the sudden death, evidently the result of the rupture of the anterior aneurism and the enormous pericardial hemorrhage; the cardiac symptoms that had troubled the patient during the last year of his life were due to the extensive interstitial myocarditis, and the peculiar outline of the area of cardiac dullness to the aneurism. They fail, however, to give an adequate explanation of the convulsive attacks, for to ascribe them to disturbances of the cerebral circulation is a pure, although perhaps, warranted assumption. The systolic murmur is difficult to understand. The valves appeared to be in all respects normal. The line of closure on the aortic leaflets was well inside the edge, the cordae tendinae sufficiently long, and the mitral leaflets not thickened. It does not seem possible that the dense resistant walls of the posterior aneurism could have permitted it to expand and receive enough blood during systole to cause a murmur, nor that the thin, inelastic walls of the anterior aneurism could have expelled enough blood during diastole to have permitted of refilling with enough force to give rise to a murmur. It is true that its cavity contained only a few clots, but the others had probably been washed out during the hemorrhage. The murmur does not appear to have been localized; and it is therefore possible that a pericardial friction sound, was, on account of the tumultuous character and the irregularity of the heart's action, mistaken for an endocardial sound.

The present case differs from the ordinary examples of cardiac aneurism by the fact that the cavities do not represent merely dilations of the ventricular wall, but communicate with the cavity of the heart by narrow channels. The situation is, however, that usually found, namely, in the region of the apex of the left ventricle, where according to Rokitansky,[1] the degenerative changes of the myocardium that cause their formation are also most common; a fact that Huchard[2] ascribes to the greater frequency of vascular alteration in this region. The pathological process that leads to the development of the myocarditis is more difficult to determine. The endocardium is practically normal and can therefore be disregarded as an etiological factor. As well as can be judged by simple inspection, the coronaries are also unaffected. This is not unimportant, for an endarteritis at, or near their orifices might cause sufficient obstruction to give rise to more or less extensive degeneration of the myocardium, although the finer arterial branches might be only slightly altered. I have more than once observed coronaries whose walls were healthy, although they were markedly contracted at their commencement. In the sclerotic areas the number of vessels appears to be diminished, and there is some thickening of the walls and lessening of the caliber, but in no case apparently complete obliteration of the lumen, in spite of the enormous hyperplasia of the connective tissue. This interstitial overgrowth, indeed, does not limit itself to the perivascular regions; but bands are often found lying between the muscle fibers themselves. It is therefore fair, I think, to exclude the chronic interstitial myocarditis secondary to arterio-sclerosis or other vascular changes. There then remain but two possibilities; either the myocarditis belongs to the group called primary, that is those in which a toxic agent expends its force principally either upon the muscle cells or their sheaths; or else it is secondary to the old chronic pericarditis. If the former, it is necessary to determine the nature of the toxic agent. Rheumatism, of course, may affect the myocardium as well as the endocardium or pericardium. The patient gives a history of vague pains that may have been rheumatic; but there is no evidence that there was ever a frank explosion of the disease. Such myocarditides, however, certainly exist and are recognixed by nearly all pathologists (Orth, Ziegler, Birch-Hirchfeld, Delafield, and Prudden, and even Huchard, who has studied so carefully changes in the myocardium secondary to arterio-sclerosis). Kelle[3] has examined particularly a number of hearts in which the myocardium was extensively degenerated, although the vessels were perfectly normal even in the midst of the lesions. In only one of these hearts was the endocardium involved. He believes that such changes may arise after various infections in other parts of the body, often when the symptoms of the latter are so slight as to escape observation.

Syphilis has also been alleged as a cause of chronic myocarditis. A number of cases have been reported by clinicians (Curschmann) that have recovered after a vigorous antispecific treatment. A few cases have been described by pathologists. In some of these the lesion was indisputable; that is to say, a distinct gumma of the heart wall was present, that occasionally, as in a case of Grassmann[4] resulted in an aneurismal dilatation of the apex.

Kockel[5] has reported as syphilitic a case that occurred in a man 55 years of age, who had had a primary lesion thirty-one years previously. For a year before death he had suffered with dyspnea. At the autopsy, the left ventricle was dilated and the myocardium permeated in all directions by dense white fibrous trabeculæ between which only a little muscular substance remained. The muscular substance had been thinned in two areas near the apex, and in both places had been forced outwards, forming a sort of aneurismal sac containing thrombi. The coronary arteries were very nearly normal. It must be admitted, however, that the actual demonstration of syphilitic changes in these lesions is lacking. Similar cases without aneurisms have been reported, in which syphilis has not been discussed as an etiological factor (Edwards). In the present instance there was in favor of syphilis the presence of the scars on the tibia, the stellate scar in the liver, and the moderate general enlargement of the lymphatic glands. The fact that the patient was unmarried is presumptive evidence that he had been exposed. On the other hand, he persistently denied infection, exhibited no other signs of the disease, and neither in the heart muscle nor the kidneys was there general diffuse endarteritis such as is usually found in these conditions. Syphilis, therefore, cannot be positively excluded, but its etiological relation to the myocarditis is extremely doubtful. The former process has been observed in tubercular myocarditis by Fontoynont.[9]

According to Grandmaison [5] chronic pericardial lesions have a tendency to cause cardiac aneurism both by giving rise to secondary sclerotic changes in the heart muscle, and by pulling upon the wall when adhesions have been formed. The latter are exceedingly common, so much so that Pepper[6] regards them as sometimes secondary. Sometimes they are associated with disease of the coronaries, as in the cases reported by Réné Marie, who expressly rejects the theory of pericardial tugging: Réné Marie and Rahe, Gesland and Griffon [7] sometimes the latter are normal as in the cases of Trenel, Gouget (loose adhesions). In the former group, of course, the vascular changes were probably the more important; in the latter, it is not impossible that the pericardial changes were the direct cause.

It is likely, in my case, that the pericardial adhesions are of long standing. That they were not the cause of the aneurism is beautifully shown by the presence of a second dilatation even larger than the first and certainly with thicker walls. The latter fact is interesting; it is possible that the thinness of the walls of the anterior aneurism was due to the continual tension to which they were subjected and the impossibility of the formation of clots on this account. In the posterior aneurism, of course, this disturbance from external causes did not exist and clots could form readily. I am not at all certain that a portion of the dense white fibrous tissue of which its wall is composed is not due to the organization of old thrombi. Bureau[8] reports that in his case such thrombi in a state of advanced organization were found intimately adherent to the endocardium lining the aneurism. In conclusion then I believe that this case represents one of the so-called idiopathic forms of endocarditis. There is not sufficient warrant for ascribing it either to vascular changes, to syphilis or to the pericarditis. Such forms are certainly rare and it is very unusual to find them leading to true aneurismal dilatations of the wall.

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November 11, 1897.

Cancer of the Stomach.

DAVID RIESMAN, M. D.

Summary.—Cancer of the pylorus (cylindric epithelioma); with dilitation of the stomach; unusual movability of the growth; hydrochloric acid present in the gastric juice; bubbling of gas through the tumor; large numbers of mastzellen in the section; slight involvement of the liver; cancerous disease of the left ovary.

The patient was a married woman, thirty-seven years of age, born in Ireland. She had enjoyed good health until a year ago, when she began to suffer from a feeling of weight in the stomach, which feeling was soon transformed into a darting pain, coming on after eating and associated with belching and sour stomach. About May, 1897, vomiting set in and the patient lost flesh rapidly. When she presented herself at the Medical Clinic of the Polyclinic Hospital (service of Dr. S. Solis-Cohen), it was found that she had a very movable tumor a little above and to the right of the umbilicus. A probable diagnosis of cancer of the pylorus was made, although omental tumor and tuberculosis of the omentum were not positively excluded. She was soon after admitted to the wards of th hospital.

On careful examination a hard mass was found a little to the right and above the umbilicus; it was very movable and could easily be pushed to the left, beyond the median line; it was also somewhat tender to the touch. Small nodules were felt in its neighborhood. The whole lower epigastric and umbilical region protruded as a rounded, hemispheric, resonant swelling, with its apex a little to the left of the umbilicus. There was a deep transverse groove passing from the costal cartilages of one side to those of the other, about 1½ inches above the umbilicus. The knee-jerk was feeble.

An examination of the gastric secretion was made for me by Dr. Garritee. *Hydrochloric acid was present* (total acidity, 40; free

HCl, 0.15 per cent.); good curdling ferment and pepsin were also found. With respect to absorbing power, it was determined that potassium iodid was absorbed and excreted in the saliva in 25 minutes; motility was impaired. The urine presented nothing significant; the temperature was generally about normal. The hemaglobin was 41 per cent. (Fleischl).

During her stay the patient complained often of cramps in the stomach, and also vomited at times large quantities of a dark, sour-smelling material; she was also constipated.

Although the symptoms clearly indicated dilatation of the stomach, I could not prove either by ordinary or by auscultatory percussion that the stomach was enlarged. On using the latter method two resonant areas separated by an irregular, dull zone (the tumor) were mapped out. As the patient did not improve, she was sent to her home, where I continued to see her. The vomiting now occurred only once in three days, but the amount then brought up was enormous, and usually filled a large wash-basin. The tumor grew larger, and on one occasion I detected bubbling of gas through it. The patient died of exhaustion after a few weeks, having emaciated to a skeleton.

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The autopsy was made on the afternoon of the day of death. On opening the abdomen almost nothing came into view except the stomach and the left lobe of the liver. The stomach was enormously dilated as well as displaced downward; the lesser curvature, near the pylorus, was on a level with the margin of the ribs, and thence extended upward into the left hypochondrium as far as the fifth rib. The greater curvature was on line four inches below the umbilicus. Transversely, the stomach extended clear across the abdomen, from left to right wall. The intestines, crowded together in the lowest part of the abdominal and in the pelvic cavity, were small in caliber; the mesenteric glands were somewhat enlarged, and appeared to be cancerous. Numerous enlarged glands were found along the greater curvature in the gastrocolic omentum, especially toward the pylorus. On opening the stomach a large growth of cartilaginous consistence was found to surround the pylorus and the adjacent region. It was covered by an ulcer, shaped somewhat like an ear, with an elevated, irregular, and overhanging edge. The pyloric orifice was diminished in caliber but not occluded. The greatest length of the growth was 70, its least 50 m. m.

The liver was small and soft; on its under surface, near the fissures, were some cancerous lymph-glands; only a few nodules, chiefly subcapsular, were found in the liver itself. The lungs were free from secondary growths. The left ovary was enlarged, and in part cystic; in its solid part, where it was whitish in color, it seemed to be the seat of cancerous change. Several pediculated cysts depended from the angle between the Fallopian tube and this ovary. A firm, walnut-sized tumor was found behind the cervix of the uterus.

Microscopic examination.—Sections of the stomach, liver and left ovary were examined. In a section of the tumor of the stomach, stained with hematoxylin, the following was observed: The mucous membrane is the seat of a marked round-cell infiltration extending from the base almost to the surface and encircling the glands. The epithelium of the tubules stains faintly and the outlines of the cells are indistinct. The tumor is a cylindric epithelioma. The stroma is abundant and fibrous in character; the alveoli are irregular—the majority have the epithelial cells arranged at the periphery, in a single layer; some are quite filled with cells, the outer ones fairly columnar, the more central ones irregular (polymorphic). The muscular layer is greatly thickened, and the cancer has penetrated between the lamina of the muscularis in the form of long, slender nests; here and there the tubular arrangement of the epithelium is preserved. There is no sharp dividing line between the mucous membrane and the tumor; the former seems to pass in to the latter. The blood-vessels are markedly congested; some contain, among the blood-corpuscles, especially near the periphery, large oval or circular bodies, about thrice the size of erythrocytes, and having the appearance of drops of colloid or fat.

A beautiful picture was obtained with thionin and eosin. The muscular tissue was pink in color, the epithelial cells bluish-purple, the red corpuscles brick red, the connective-tissue cells and leukocytes blue. The most interesting feature in these sections was the large number of "mastzellen." These were found especially in the muscular tissue—in the inter-muscular planes, and in areas of round-cell infiltration lying in these spaces; also in the neighborhood of the blood-vessels, and about the nests invading the muscular tissue. None were found in the mucous membrane proper, or in the part of the cancer contiguous to it. The cells were large, and usually contained a single nucleus staining darkly. The protoplasm was completely filled with granules looking exactly like micrococci, and taking a dark, reddish-purple stain. In many cells the nucleus was hidden by the granules.

The cells were generally oval or spindle-shaped; in some instances one or two processes crowded with granules projected from them. In certain areas it seemed as if the granules had been discharged from the cells, lying free near them. In hematoxylin sections very few of these cells could be made out.

The thionin also stained the bacteria present; one tubule seemed to be full of cocci and bacilli.

The liver-sections, where a cancer-nodule was embraced in them, showed the features of the primary growth. In the left ovary many hyaline blood-vessels were found, some almost obliterated by thrombo-arteritis; there were also cancer-nests similar to those in the stomach and liver.

With respect to the cancer of the ovary, the question suggests itself—was it secondary to the cancer of the stomach, or could it have been a coexistent primary growth? The evident similarity in microscopic appearance favors the former view.

DISCUSSION.

DR. STENGEL—Tumors of the stomach are often very movable, especially when the organ is dilated, for in such cases the pylorus may be torn loose and dragged downward. Under such circumstances the condition may closely simulate an omental tumor. Another dogmatic teaching in text-books is that tumors of the stomach do not move during respiration; such is not the case; they often move, either on account of adhesions or because of the movements transmitted through the diaphragm. In connection with the question of the mastcells, I wish to insist upon the frequency of fine and coarse basophilic granulations in human blood. Canon had first shown that fully 10 per cent. of the leukocytes might present such granulations, and I have noted the same thing. Westphal showed that they were present in large numbers in the blood of starving animals, of hibernating frogs, and in conditions of emaciations in general. I believe that they are to be viewed as evidences of degenerative processes, perhaps as themselves degenerated. Their nature is entirely obscure.

DR. TAYLOR.—I have for some time made it a practice to stain sections of all tumors with thionin or neutral-red, since which I have not seen a single carcinoma which did not contain mastcells, often to an enormous extent. I have seen them as the sole cellular elements of a myxomatous tumor of the bladder, and have seen several tumors from the nose which contained masses of these cells imbedded in a homogeneous substance which took acid stains. In many tumors it has seemed that they were most abundant about those areas of the growth where karyorrhexis and karyolysis were most frequent. Since the supposition that the granules are fatty has been disproved, no evidence has been adduced to show that they are nutritional, and it certainly seems likely that they are degenerative.

DR. ABBOTT referred to the occurrence of the granules about areas of experimental edema and necrosis, and alluded further to their presence in the testicle of the guinea-pig.

DR. GARRITEE referred to three cases of gastric neoplasm in which the gastric contents contained a high degree of acidity with abundance of free HCl; on operation two were determined to be cancer, while the third was a fibrous overgrowth about the site of an old ulcer.

November 11, 1897.

Melanotic Sarcoma of the Liver.

J. H. MUSSER, M.D.

This huge liver was removed from the body of a patient aged forty-two years, who presented during life the following symptoms which led to the diagnosis of melanotic sarcoma of the liver. In July of the present year he noticed some distension of the abdomen, and at the same time incapacity for exertion. He began to lose flesh, and from this time loss of flesh and strength was continuous. On admission to the hospital in August, 1897, the physical signs of enlargement of the liver were found. The surface was smooth, the left lobe could be readily distinguished from the right by a distinct notch. One nodule was felt at the margin of the right lobe above the ileum. Two weeks before death the upper border of the liver began at the fourth interspace anteriorly, and extended below the umbillicus and its area increased rapidly before death. The upper two-thirds of the right half of the abdomen and the upper left quadrant was occupied by the mass. There were no other events in the course of the disease except the occurrence of ascites two weeks, and of slight jaundice one week before death. There was pronounced anemia, the blood count being as follows: Red cells, 3,360,000, leucocytes 14,980 per c. m. m., hemoglobin 52 per cent Fleisch. Examination of the other abdominal organs yielded no information of interest. With the above-mentioned history and physical signs, and the history of the removal of the left eye in August, 1895, at the Wills Eye Hospital, for melanotic sarcoma, the diagnosis was made. The duration since the operation was two years and two months; duration of illness after hepatic symptoms began, four months.

At the autopsy this splendid specimen, weighing 17½ pounds, was found. There were also secondary growths in the lungs, retroperitoneal and mediastinal glands and on the right fifth rib internally. DR. RIESMAN has reported the result of microscopic examination: round-celled sarcoma, with marked melanosis and dense fibrous hyperplasia.

October 28, 1897.

Tuberculosis of the Kidney.

J. M. SWAN, M. D.

The subject from whom this specimen was taken was a female aged thirty-five years, who died in the Episcopal Hospital. The woman was admitted with pulmonary tuberculosis and died soon afterwards. At the time of admission the patient was in the sixth month of pregnancy. At the autopsy the lung was found tuberculous, and, in addition, an extremely interesting lesion was discovered in the right kidney. It was enlarged and the perirenal fat was nearly entirely absorbed. The capsule was much thickened and was quite adherent. The upper end of the ureter and the entire pelvis were dilated and contained fluid. On section the organ was found to be studded with small tubercles. The distribution of these lesions was such as to indicate an hematogenous infection. Although the specimen was not injected it seems that there can be little doubt but that the tubercles developed along the course of the blood-vessels both in the cortex and in the medulla. It would be interesting to know why, in an hematogenous infection, such as this, one kidney should be healthy and the other so extensively diseased. The left kidney was the seat of a chronic parenchymatous nephritis, but presented no tubercles. The fetus was a male and, to all appearances, normal.

DISCUSSION.

DR. STEELE.—I wish to call attention to Meyer's work in this connection. He found tubercle bacilli in foci inside the urinary tubules without solution of continuity of the membrane of the tubule. This he terms a secretion-tuberculosis.

October 28, 1897.

Specimen of Internal Strangulation of the Small Bowel by a Thread-like Band.

JOHN B. ROBERTS, M. D.

The specimen here presented was obtained from a man of middle age who had suffered from constipation for two weeks and obstruction for five days. When I saw him his temperature was below 96°, his abdomen greatly distended, and he was vomiting material stercoraceous in character. Preparations were made for immediate exploratory abdominal incision. During the hour that was required for sterilizing instruments and preparing for operation the man's extremities became cold, his face was covered with a clammy sweat and he died just as etherization was about to be begun.

The autopsy, made within an hour, showed no evidence of peritonitis, but great distension of the small intestine down to a point about sixteen inches above the iliocæcal valve. The intestine from this point to the cæcum was collapsed and deeply congested.

Investigation showed that this loop of bowel was encircled by a thin cord of tissue, looking very like white string about the size used for tying grocers' parcels. This band or cord, which was 13 c. m. long, was attached at one end to the parietal peritoneum on the front of the abdominal wall and at the other to the mesentery above the upper portion of the constricted bowel. From another portion of bowel hung a pedunculated mass of tissue, 4½ c. m. long which looked like several lymphatic glands held together by connective tissue. The thread-like band previously mentioned ran through a little opening in this appendage, as a cord is run through a pulley. The appendage was attached to the intestine opposite the mesentery, but had no opening in it as would be expected if it were a Meckel's diverticulum. When first I saw this structure, its shape and size caused me to suspect that it might be the vermiform appendix shortened and twisted as a result of old inflammation. I soon however, found that this was not true. It was not attached to the cæcum and I subsequently found the appendix which it was perfectly normal. The constriction of the bowel by the thin white round cord was so tight that the portion of intestine toward the cæcum had a dark line, at the bottom of the groove made by the cord, similar to the congested gangrenous line often seen in tightly strangulated hernias.

It seems to me probable that the man had had in earlier years an adhesion of the intestine to the anterior abdominal wall and that the attachment had gradually become stretched until a long thin cord had resulted. There was no evidence of chronic inflammation or adhesion noticed, except the pedunculated mass consisting of lymphatic glands and the strangulating cord. It was difficult to tell what part the thicker mass played in the fatal intestinal constriction. That the long thread was the essential factor was, I think, evident from the deep groove in the intestinal wall noticed at the lower part of the strangulated gut. A loop had worked its way under this band which had its freedom of movement limited by its attachment to the pedunculated mass at the pulley-like opening mentioned.

Operation would apparently have been very easy if undertaken in time. The great distension when the patient was seen by me would have probably compelled me to make openings in the intestine to evacuate gas and feces; but I could then have readily examined the seat of obstruction and relieved it. The fact that no peritonitis had occurred and that the constricted portion of bowel was not gangrenous, though deeply congested, seems to indicate that the operation would have been successful, if the fatal collapse of the patient had not occurred before opportunity was given for operative work. *October 28, 1897.*

Cerebral Hemorrhage.

J. H. MUSSER, M. D.

The specimens are from a male nurse, *act* 43. Married. He used tobacco in moderation and beer to excess. Always well. No syphilis. His parents died of old age. The illness began last winter with dyspnea on exertion, and later he noted swelling of the feet. In the spring he was unable to work, but improved and felt well during the summer. In September dyspnea and swelling of feet returned and since then he has had asthma and palpitation. The temporals were prominent, and the carotids throbbed. There was high arterial tension and hypertrophy of the heart. When first seen there was gallop rhythm, and a slight murmur was heard at the apex, which was in the sixth interspace outside of the nipple-line. Later, the gallop rhythm and murmur lessened and with that come relief to dyspnea. The heart's impulse was violent and *knocking*, strong and heaving, without rhythm or murmur, but with a loud accentuated aortic second sound and occasionally a splash suggesting incompetent aortic valves. The patient was much improved by nitro-glycerine, Tr. strophanthus and Hoffman's anodyne. The urine presented the evidence of chronic parenchymatous neplixitis. While on the street he was suddenly seized with loss of power in the left leg and arm, became confused and dazed, and had aphasia. He was able to indicate the card-case in which his address was found, and able to pay the cab man when he arrived home. In the course of an hour sudden unconsciousness came on, and death occurred in about an hour after the onset of the coma.

Diagnosis.—Chronic parenchymatous nephritis—hypertrophy of heart—death from apoplexy.

Post-mortem.—There was general arterial sclerosis. The abdominal cavity was relatively normal. The liver was somewhat cyanotic but presented no signs of cirrhosis. The spleen was slightly enlarged and congested. The left kidney measured 9 x 2 c. m., and weighed 130 grms., the right 10 x 4 c. m., and weighed 140 grms. The capsule strips readily, the surface is pale and yellow. The organs are granular and scarred with a few cysts, in places soft, in others hard. On section the cortex is soft and yellow though in places are areas of congestion. The pyramids are not congested. The heart is much enlarged, its weight 736 grms. The left ventricle measures 28 m. m., the right 10 m.m., in thickness. There are a few spots of atheroma upon the aortic valves, but the aorta above the valves is excessively atheromatous and presents many calcareous plates.

The coronar arteries are unusually healthy, presenting only a few scattered spots of sclerosis. The myocardium presents no scars or evidences of fatty degeneration. There are a few spots of atheroma at the base of the anterior leaflet of the mitral valve, which appears competent and not dilated. The pulmonary and tricuspid valves are healthy and not dilated. The cerebral and basilar arteries are very much sclerosed, and stand out like tubes passing into the fissures. Numerous spots of atheroma are seen. A very large hemorrhage is seen in the right lateral ventricle, and it has passed through into the left ventricle, in which the blood is in a slightly fluid state; the primary hemorrhage is into left internal capsule. There are miliary aneurisms on the basal branches, throughout both internal capsules.

DISCUSSION.

DR. SPILLER inquired whether the patient had had any convulsions after the stroke. It was a general teaching that intra-ventricular hemorrhage might likely be accompanied by convulsions, which have been held to be due to the irritation of the motor fibers by the clot; he believed that such a clot would more likely press upon the tracts sufficiently to cause paralysis instead of spasms. Dr. Arnold recalled a case with hemorrhage into the right ventricle, in which marked and repeated convulsions occurred.

November 11, 1897.

Abscess of Lung.

JOSEPH SAILER, M. D.

It appears from the clinical history of this case that the process began as an acute croupous pneumonia in the lower lobe of the left lung. This continued for about three weeks, when there was a sudden rise of temperature which, for the next ten days, was hectic in type. Toward the end, the apices of both lungs also became consolidated. There was an abundant, purulent expectoration, containing a great variety of micro-organisms, but no tubercle bacilli. The temperature at death rose to 107°. At the autopsy the specimens were found as here presented. The left lung was densely adherent to the thoracic wall and the diaphragm, the pleural cavity being totally obliterated. Its lower portion contains a single huge abscess that replaces the whole of the lower lobe. The upper lobe is consolidated. Upon section, grayish, and upon pressure a purulent liquid is exuded. The upper lobe of the right lung is in a similar condition. The spleen was enlarged and friable. The kidneys were slightly sclerotic, and in the left were several abscesses, the larger ones being as large as 3 c. m. in diameter. The right kidney was about two-thirds the size of the left, and contained several small abscesses. Smear preparations of the pus from the pulmonary abscess, the purulent pneumonia, and the abscesses in the kidneys showed an almost pure culture of a short, thick bacilli, whose exact nature has not yet been determined.

DISCUSSION.

DR. ABBOTT commented upon the rarity of such extensive visceral pyemia in recent years, and alluded to the fact that before the introduction of antisepsis such conditions were of frequent occurrence.

November 11, 1897.

CARD SPECIMENS.

Carbolic Acid Poisoning.

H. W. CATTELL, M. D.

The esophagus, stomach and kidneys had just been removed from a woman who died in convulsions one hour and fifteen minutes after taking an unknown quantity of pure carbolic acid. He mentioned from personal observation: (1) That the odor of carbolic acid can sometimes be noted in the kidney, even when it cannot be detected in the stomach. (2) That carbolic acid, owing to the ease with which it can be obtained, to the promptness and certainty of its action, and to its anesthetic properties, is one of the most popular means for committing suicide. (3) That the esophagus presents in these cases a silvery lustre which darkens on exposure to the air. (4) That in the passage of the tube for washing out the stomach, the membrane formed by the caustic action of the drug, is often pushed downward from the upper portion of the esophagus and impacted in the lower part near its entrance into the stomach.

October 14, 1897.

Dissecting Aneurysm of Aorta.

J. M. SWAN, M. D.

A dissecting aneurysm four inches long, running between the media and adventitia, from just below the sinus of Valsalva. The sac, which was partly filled with clotted blood, lay directly behind the pericardium. The heart and aorta were enlarged, and the aorta very atheromatous.

October 28, 1897.

Gastric Carcinoma.

E. R. SCHREINER, M. D.

The primary tumor was a large soft mass around the pylorus, about the size of the fist. In the liver, pancreas and adjacent lymph-glands were nodes with pus-like softening in the central areas. There was also an abscess in the right calf, whether metastatic in nature, however, could not be microscopically determined.

October 28, 1897.

Gastric Carcinoma.

J. B. ARNOLD, M. D.

The growth was not larger than a walnut, and situated just above the pylorus along the upper curvature. The metastasis to the liver was extensive, and in marked contrast to the size of the primary tumor.

October 28, 1897.

Proceedings
of the
Pathological Society of Philadelphia.

JANUARY 15, 1898. NEW SERIES, VOL. I, NO. 3

TABLE OF CONTENTS.

POTTS, Tumor of the Spinal Cord.—**SAILER**, Changes in the Central Nervous System after Aseptic Injury.—**STEELE**, A Review of the Literature of Koch's Tuberculin R.—**DALAND**, Appendix Vermiformis with Pin in Situ.—**ANDERS** and **MCFARLAND**, Pneumothorax with Discovered Perforation.—**RIESMAN**, Congenital Inguinal Hernia.—**OSTHEIMER**, Gangrenous Carcinoma of the Stomach.—Card Specimens: **RIESMAN**, Gangrene of Lung; Tumor of Infundibulum; Anomaly of the Basilary Artery.—**OSTHEIMER**, Slides Showing Basophile Cells.

Tumor of the Spinal Dura Mater.

C. S. POTTS, M. D.

The subject was a boy *aet* sixteen years. The family history was negative. The left leg of the patient was amputated about eighteen months ago on account of a sarcoma involving the knee-joint.

About one year afterward, in July, 1897, he noticed that the right leg was becoming weak. Three days after the weakness was first noticed the leg would not bear his weight, but he could still move it to some extent. During this time the leg felt numb, and tingling sensations were experienced. These symptoms only lasted about a week. In two weeks from the time of the onset of symptoms, power of motion was completely lost, but involuntary contractions of the muscles manifested themselves. During this time he had no pain.

The patient was examined by me on August 30, 1897, and the following condition of affairs was noted: There was a complete loss of power in the right leg and in the stump of the left; passive motion elicited slight rigidity of the muscles; there was muscular atrophy, but the electrical reactions were normal; a slight plantar reflex could be elicited by violent irritation, the cremasteric reflex was present, the abdominal reflex was absent. Ankle clonus was marked and the knee jerk much increased; irritation of the skin of the leg, as by pricking it with a pin, caused involuntary muscular movements; these movements also took place spontaneously, and at times were very annoying. The sphincters of both bladder and rectum acted normally. Tactile sensibility was lost in the leg and stump, and on the abdomen to a point about one inch above the umbilicus, and the area extended around the body to the back, where the limit was about two inches higher. The pain-sense was abolished in the same area, excepting that over the right buttock and inside of the right thigh it was not entirely absent, and in these two situations a strong, rapid, interrupted faradic current could be felt. The temperature-sense was lost in the same area. Muscular sense, as determined by the knowledge of the position in which the leg was placed, was good. There was no hyperæsthesia, —nor pain. He complained of no trophic or vasomotor symptoms.

In October he began to pass urine involuntarily at night, but could hold it for a short time when awake. Ankle clonus became less marked and sensation appeared to be somewhat better, for when pricked with a pin he knew that something touched him, and compared the sensation to being touched with the head of the pin. The conditions of sensation and the deep reflexes varied in this way, being at times better, at others worse for several weeks.

Two weeks later he complained of pain between the shoulders after sitting up for a while. Examination revealed no cause for it, and it disappeared in a few days. Late in October he began to pass urine involuntarily while awake, but knew when his bladder was full.

The level of anæsthesia extended upwards until it reached a point about two inches below the ensiform cartilage in front, and to a point a short distance above this behind. It completely involved all parts below this level, touch, pain and temperature senses being affected. He next lost the consciousness of when his bladder was full, and of urination, but there was no dribbling. A small amount of residual urine was removed when he was catheterized. These two symptoms remained constant until death.

Late in November he again complained of a dull pain between the shoulder blades, aggravated by sitting up. On the first day of its appearance it extended into the right axilla, but this soon disappeared. The pain was never severe enough to cause him to lose sleep. About this time a bed sore appeared on the left buttock, just to the left of and a little above the anus.

A week late another decubitus made its appearance over the right trochanter, but under treatment it healed up, while the first one made no progress towards healing and was about four inches long, and two wide.

From this time on the patient rapidly grew weaker. The muscles of the right leg, already atrophied from disuse, wasted until below the knee hardly any muscle could be felt. Three weeks before death he declined to sit up in his chair, preferring to remain in bed, and continued bedridden until death, lying on his back with the leg rotated outwards, the thigh flexed upon the abdomen and the leg flexed upon the thigh. The involuntary muscular contractions were from this date much diminished in frequency and force.

He complained of paroxysms of a dry hacking cough, but examination showed the lungs to be in good condition.

On December 13, he suddenly became extremely weak. The extremities were cold, pulse almost impalpable, the breathing rapid and labored; he also complained of nausea. He reacted and in three days was as usual; on that date examination showed that the plantar reflex, ankle clonus and knee jerk had disappeared.

On December 20 he had a similar attack of collapse; examination of the lungs revealed the presence of dry râles. He did not react and died at 5 p. m. apparently of respiratory failure.

Dr. Sailer reported the results of the autopsy as follows: Body of greatly emaciated boy. The left leg had been amputated, leaving the upper third of the thigh. There was a large sloughing bed sore over the sacrum; rigor mortis and post mortem hypostasis were present. Upon making the incision over the spinal column, the subcutaneous tissues were found to be markedly œdematous; the blood was still fluid and flowed freely from the cut. Over the lower part of the dorsal region the muscles were somewhat elevated and filled with masses that resembled calcareous infiltration. These masses were found upon further dissection to be continuous with the substance of the vertebræ. The spinal column showed a lateral curvature with the convexity to the right. In the lower part of the dorsal region the lamellæ were softened. In the other portions the spinal column appeared to be normal. The spinal canal in the middle dorsal region was considerably narrowed by the growth of this hyperplastic and softened bone. The spinal cord was reduced to about one-half its normal diameter. Upon removing the cord an irregular mass was observed upon the outer side of the dura, anteriorly, extending from the ninth to the eleventh rib. In the upper dorsal region a small hemorrhagic cyst was observed upon the posterior surface of the dura. The dura mater was not adherent to the spinal canal nor to the arachnoid. The pia and arachnoid showed no pathological alterations. Section through the cervical region revealed the existence of degeneration in the column of Goll; in the upper region, of the columns of Goll and Burdock. In the lumbar region the pyramidal columns were swollen, soft and paler than the rest of the white matter. Microscopical studies have not yet been made. The brain and viscera could not be examined.

DISCUSSION.

DR. DACOSTA.—Following the amputation of the leg in this case, very unusual phenomena occurred in the stump. The muscles of the thigh were seized with choreiform contractions and the flaps were torn loose. Reamputation was performed, again the contractions tore the flaps loose; a second reamputation was done, following which the stump slowly healed. Could these most unusual movements have been the result of a cord lesion; and is it possible that the cord lesion antedated the femoral growth instead of being a metastasis from it?

DR. SPILLER.—The explanation of the absence of pain in this case probably lies in the size of the growth and its location anterior to the cord. The friable tissue of the vertebræ probably did not cause much pressure on the posterior roots. Cases of tumor within the spinal canal without pain have been reported (Pearce Bailey). The presence or absence of pain has been considered of value in diagnosticating between malignant growths of the spinal canal and Potts' disease in its early stages. It is a symptom upon which we cannot depend to any great extent.

DR. SAILER.—If the tumor-like hyperplasia of the vertebræ in the middle dorsal region had pressed upon the posterior cord so as to have shut off the afferent impulses, painlessness would have resulted even though the posterior roots below this point had been involved.

DR. POTTS.—The suggestion that the cord lesion antedated the growth in the leg may receive confirmation from the fact that the symptoms progressed so rapidly after they first manifested themselves, much more so, it seemed to me, than is usually the case in compression myelitis. The symptoms might first have manifested themselves in the left leg, the muscular contractions spoken of by Dr. DaCosta being due to anterior nerve root irritation, and paralytic symptoms that might follow would naturally not have been noticed in a short stump.

J. Mitchell Clarke has also reported in Brain a case of tumor of the cord in which there was no pain. This growth was an endothelioma and was anterior.

December 23, 1897.

Changes in the Central Nervous System after Aseptic Injury.

(FROM THE PEPPER LABORATORY OF CLINICAL MEDICINE.)

JOSEPH SAILER, M. D.

In the early part of 1897 Tedeschi[1] published a very interesting paper upon the regeneration of tissue in the central nervous system. He caused injuries aseptically in various ways, as with a red hot needle, and Paquelin's cautery; he removed with the knife a greater or less portion of the cerebral hemisphere; and he introduced foreign bodies into the substance of the brain. Of the latter, he obtained particularly good results with paraffin, which was light enough not to sink through the soft brain substance, had no chemical action upon the nervous tissue, was easily rendered aseptic, was soft in consistency, and above all, could be cut with the nervous tissue, without injuring the knife or disturbing its relation to the injured and proliferating neuroglia. His results, particularly after the application of the paraffin technique, were exceedingly interesting. In the neighboring parts he found proliferation of the neuroglia cells, the endothelial cells of the blood-vessels, and the ganglion cells. Particularly in the two last, he found typical karyokinetic figures, and he concludes from this, that there is no reason to believe that these cells cannot undergo division. The scars that resulted were composed chiefly of neuroglia, in the midst of which a few ganglion cells and nerve fibres could be found.

Marinesco,[8] in a recent paper upon the effects of various pathogenic agencies upon nerve cells, describes the changes produced by traumatism as follows: the lesion may be divided into a central necrotic zone and a peripheral reaction zone. In the former, the nervous tissue is converted into a uniform mass with dilated vessels and diffusely staining nerve cells. In the peripheral zone there are leukocytes, many presenting karyokinetic figures. From the third to the eighth day after injury, karyokinetic figures can also be observed in the nerve cells, but as it is impossible to detect any proliferation of the latter he holds that regeneration of the central nervous system after injury does not occur, a conclusion that would only be warranted, if the karyokinetic figures exhibited degenerative alterations, such as those described by Galleotti, but this does not appear to have been the case, at least Marinesco makes no allusion to such appearances.

It occurred to me that it would be interesting to study the results of simple aseptic laceration of the brain. For this purpose I took a cat and, having anesthetized it, aseptically exposed the skull, and bored through it with a small trephine about 2 mm. in diameter. Through this opening I plunged a platinum wire that had been carefully sterilized in a Bunsen flame, and moved it gently to and fro. After recovering from the anesthesia, the cat showed some disturbance of motion in the left hind leg for a few hours, but this soon disappeared. It seemed very quiet and refused nourishment; not even appearing to be thirsty. At the end of 72 hours I killed it, removed the brain, and cut it into small pieces, that were placed in 95 per cent alcohol. The macroscopic lesions consisted of a slight protrusion of the brain substance through the trephine opening and a considerable extravasation of blood at the site of injury. The latter I had not anticipated, and considered it unfortunate, because, to a certain extent, it must have acted as a foreign body. The tissue was imbedded in celloidin and sections made that were stained with Nissl's methylen blue solution, with thionin solution, with hæmatoxylin and eosin, with thionin and fuchsin, and by Rosin's and Van Giesson's methods. With methylen blue and thionin, the results were approximately the same.

The brain not directly involved in the injury appeared to be normal; this appearance was controlled by sections from the opposite hemisphere. The path of the platinum wire appeared to be filled with blood pigment arranged in amorphous brownish masses, somewhat rectangular in outline. In the immediate neighborhood of the lacerated tissue there appeared to be no increase in the number of neuroglia cells, the tissue not staining at all. There was no accumulation of leukocytes. Just beyond this zone, capillaries could be seen pushing their way toward it, which were composed of slender loops of endothelial cells, some of whose nuclei were undergoing karyokinetic changes. In this area the neuroglia cells were slightly more numerous than in the normal tissue. Such of the ganglion cells as could be seen were extensively degenerated, the majority being quite irregular in outline and staining diffusely and faintly. The processes appeared to be broken off, were thicker, and somewhat varicose. Beyond this region, the tissue showed less and less involvement; the neuroglia cells rapidly becoming normal in number; and the ganglion cells staining as did the cells in the healthy side. When acid fuchsin and eosin were used in the stain, the neuroglia fibres became quite distinct. In the normal areas they formed a delicate fine-meshed reticulum, that, with the low power, appeared to be homogeneous. In the immediate vicinity of the clot they stained more intensely, the fibres being thicker and the meshes much larger, so that even with low powers the injured area could be seen filled with a course mesh work of stout threads. This change in the neuroglia ceased rather abruptly at the commencement of the zone of proliferated endothelial cells, that is to say, the mesh work became suddenly finer but not as fine as that of the normal tissue, into which it gradually faded.

The changes as observed in the sections stained by Van Giesson's method and by hæmatoxylin and eosin, contributed no additional information. When Rosin's stain was employed, it could be seen that the newly-formed neuroglia tissue took the red with distinctly more intensity than the normal neuroglia, in fact took it to the entire exclusion of the orange. It seems reasonable to conclude from these preparations, that, after aseptic laceration of the central nervous tissue, there is a marked and prompt increase in the neuroglia tissue to take the place of the destroyed tissue. This increase is chiefly manifested by thickening of the fibres and not by the direct proliferation of the neuroglia cells; but a moderate proliferation of the neuroglia cells occurs just beyond the area of direct injury and it is possible that it is from these that the fibres project. There is also regeneration of the vascular supply in exactly the same way that this regeneration occurs in any other tissue. The ganglion cells in the area of destruction degenerate completely. It does not seem possible that those that I observed could ever have resumed their functional activity. Neither Goldscheider and Flatau[3] nor Kempner and Pollak[4] have described such extensive alterations in animals, poisoned by toxins, that were still susceptible of complete cure by anti-toxin treatment. In none of the ganglion cells in the neighborhood were any signs whatever of karyokinesis observed, but it is probable that the animal was not kept long enough alive. Tedeschi is certainly qualified to recognize karyokinetic figures and furthermore, Buchholz[5] has observed them in the ganglion cells of young dogs and rabbits, although he admits that they were not as typical as the figures found in the endothelial cells of the blood-vessels and the ependymal cells of the ventricles and the central canal of the cord, and that they were found only in cells possessing an undeveloped structure. Marinesco's observation of karyokinesis in leukocytes is very unusual, and the fact that both Tedeschi and myself observed these changes in endothelial cells leads me to believe that in this statement he is in error. Throughout the whole of the affected hemisphere pyknomorphous cells were more common than on the other side. This only confirms the statement of Nissl[6] that they are more common in the neighborhood of pathological lesions, although this observer seems to regard them as artefacts or physiological conditions. Personally I cannot entirely agree with this view, for, in a case in which I injected some anthrax culture into the brain of a guinea pig, the animal dying three days later, I found anthrax bacilli apparently in the brain tissue and not limited to the capillaries, and an enormous number of ganglion cells that stained deeply and diffusely. The results of this experiment can therefore be looked upon as essentially a confirmation of Tedeschi's work, the only point of difference being the absence in the brain of my cat, of any signs of regeneration in the ganglion cells. I purpose, however, to repeat this experiment, and to keep the animal alive if possible, for several weeks, and hope that I shall then be able also to confirm this point.

REFERENCES: (1) Tedeschi.—Ziegler's Beiträge, vol. xxi, p. 43, 1897. (2) Marinesco.—Twelfth International Medical Congress at Moscow. Fortschritte der Medicin, October 15, 1897. (3) Goldscheider und Flatau.—Fortschritte der Medicin, April 1, 1897. (4) Kempner und Pollak.—Deutsche med. Wochenschrift, No. 32, 1897. (5) Buchholz.—Neurologisches Centralblatt, p. 140; 1890. (6) Nissl.—Neurologische Centralblatt, No. 19; 1894.

DISCUSSION.

DR. SPILLER.—The neurogliar overgrowth present in these sections—consisting almost entirely of fibres without cells—is very remarkable when the duration of these lesions—three days—is considered. The staining methods of Weigert and Mallory for the neuroglia seem to demonstrate, that there is a separation of the neurogliar fibres from the cells. Unfortunately, these methods can only be successfully employed in the study of human nervous tissue, but similar experiments to those described by Dr. Sailer are sometimes performed on the human brain, as in surgical operations, and opportunity is afforded for the study of the development of the gliar fibres. E. W. Taylor, in a recent paper, states that the gliar fibres are probably at first part of the cell and become separated from it in their later development.

December 23, 1897.

A Review of the Literature of Koch's Tuberculin R.

J. DUTTON STEELE, M. D.

It is now over half a year since Koch's original article appeared in which he announced the discovery of tuberculin R, and his experiments by which he showed the possibility of producing by its use immunity in the guinea-pig against the tubercle bacillus and its products. Many articles have since appeared, and we may conclude that the first period of this literature is finished; by this is meant that in which the results of numerous series of observations are recorded. From these observations conclusions may be drawn as to the immediate effects of tuberculin R upon the general system and upon various tubercle foci already existing in the body.

H. Buchner claims for his brother Edward Büchner priority in the idea of mechanically breaking up tubercle bacilli for the purpose of facilitating their absorption. He published his article with an account of his experiments in 1893. He states that he is able to pulverize the tubercle bacilli by grinding them with sand, and thus avoid the danger which is incurred when they are powered in a dry state. The material is obtained from a laboratory in its concentrated form, and diluted to a proper strength by glycerin or a normal salt solution. In general, the commencing dose was $\frac{1}{500}$ of a milligramme, which Koch says should be doubled every second or third day. Most observers, however, have found that this is too severe and increase the dose much more slowly and at intervals determined by the amount of reaction. The maximum of 20 milligrammes is seldom reached, because of excessive reaction or lack of opportunity to finish the cure. Bussenius gave one patient 40 milligrammes and several others 20 milligrammes as a maximum dose. The maximum in other cases varied from 6 milligrammes to 4 milligrammes. Bussenius reports four cases who finished the "cure," that is reached the maximum dose of 20 milligrammes. These cases received large doses of the old tuberculin without reaction. Von Neucki, v. Maczewski and v. Logucki found in freshly opened bottles of tuberculin R live pneumococci, streptococci, staphylococci and several varieties of moulds and yeasts; they also found a few tubercle bacilli in two cases. The specimens obtained in bottles with cork stoppers were much more likely to be contaminated. Trudeau and Baldwin found that all of twenty-eight bottles with cork stoppers and seven out of forty-eight bottles with glass stoppers contained yeast-like growths, which were not, however, pathogenic to guinea-pigs. One or two bottles contained living tubercle bacilli. Bussenius, however, examined many specimens, and found them sterile and free from tubercle bacilli.

The injections are given with a hypodermic syringe under strict aseptic directions. In a majority of cases there is decided local reaction about the point of injection. But few abscesses are reported, but in several series such occurred, in one of which the pus was examined for tubercle bacilli without result (Rumpf). Several observers report cases in which an old point of injection became inflamed after a subsequent injection in an entirely different part of the body. Infiltration and redness lasted but a few days. Urticaria was observed in one case by Bruno-Leick, and erythema in two cases. The different outputs of the material show great difference of strength. Bussenius attributes much of the difficulty of preventing excessive reaction to this fact. Thus, a man may reach a large dose without reaction and react strongly at the next dose. The output of June 11, 1897, seemed particularly toxic (Bussenius, Van Hoorn and Wörner). Bussenius and Baudach complain of the variability of the same specimen in the same patient. It is difficult to explain the irregularity and variability of the reaction upon any other ground. Kraatzer thinks that there is an idiosyncrasy to T R.

Koch says that there should be no reaction greater than .5° C. Most observers, however, report much greater rises in temperature. The highest variation occurred in a case of Bussenius's series, and the patient showed a temperature of 40° C. Bruno-Leick doubts whether much of this fever, especially when the injection is given in the evening, is not due to the course of the disease. Symptoms of general nervous disturbance, headache, dizziness, chilliness, pain in the back and limbs, occur usually at the same time as the fever, but may not do so. Reaction in the urinary tract, as hematuria and albuminuria, is usually absent. Rumpf reports a case in which but trifling reaction occurred and a dose of 20 milligrammes was reached without reaction; however, four days later intermittent fever commenced, with an evening temperature of 39° C. The point of the injection became very much inflamed and an abscess formed. The pus was examined for tubercle bacilli without result. Rumpf considers this to be an evidence of accumulative action, but it would seem without good reason; it is much more likely that there was a pyogenic inflammation through contamination of the material or imperfect asepsis, and that the febrile process was dependent upon the abscess. The lymph glands in the neighborhood of the injection occasionally enlarged; the enlargement, however, was temporary, and no case was reported in which they broke down.

Koch's directions as to the character of cases to be chosen have been generally followed. Such cases are those in which the range of the evening temperature does not exceed 38° C., and in which there is no evidence of infection by the pyogenic micro-organisms.

All observers agree that it is too soon to state positively the effect of the treatment upon tubercular foci in the lungs. As Koch has asserted, the sputum, the râles, and number of bacilli usually increased at first, and then diminished. The physical signs, as a rule, remained about the same, although a few cases appeared to be improved and a few grew worse.

Kraatzer reports a series of eight cases, in five of which there was decided diminution in dulness, râles, and bronchial breathing, but no improvement in one and loss in two. He brings forward nothing to prove that the change was due to tuberculin R.

In lupus injections gave almost invariably good results. Doutrelpont reports fifteen cases and Bussenius three, in all of which there was marked improvement during the course of injection. Several of them, however, had been previously cured by the old tuberculin, and it remains to be seen whether the effect of the new remedy is any more permanent.

Herzfeld reports seven cases of laryngeal tuberculosis treated by T R. Two improved, four grew worse, and one remained unchanged. Bussenius states that the fears of a reactionary edema of a laryngeal ulcer sufficient to cause obstruction are unfounded.

Müller reports two cases of otitis media treated by tuberculin R. One was a patient with tuberculosis of the lung and tubercular otitis of three months' standing. After four months' treatment with T R, miliary tubercles appeared in the tympanum of the sound ear, and the ear commenced to discharge; tubercle bacilli were found in the pus. He discusses the responsibility of the T R for the condition, and suggests that it may have caused greater activity of the tubercle bacilli in the tubercular process in the lung and free entrance into the blood-stream. However, as the lining of the middle ear was simultaneously affected, he considers it much more likely that the source of the inflammation was the entrance of the bacilli through the eustachian tube during coughing. He mentions the possibility of the injected material carrying the causes of the inflammation. In neither of the cases did benefit result from the treatment. Slawyk reports two cases of tuberculosis in children treated with T R, and concludes that for them the dose must be very small, and is to be increased much more cautiously than in adults; the maximum should never exceed 2 milligrammes.

The following conclusions may be drawn:

1. The new preparation, if uncontaminated, does not seem to be more harmful than the old tuberculin if very carefully given. The dosage suggested by Koch is probably too severe. Much is left to be desired in the preparation of the material. In its present form it is usually contaminated. The greatest element of danger is the possibility of the presence of living tubercle bacilli. It may also contain streptococci, diplococci, staphylococci and various saprophytic bacteria. Certain outputs of the substance are clearly stronger than others and more likely to cause serious reaction.

2. The injections are accompanied by much discomfort to the individual. The point of entrance of the needle usually becomes the seat of considerable inflammatory reaction and occasionally of abscess formation. Much of this may be accounted for by the contamination of the preparation or faulty asepsis in its administration; but, even in the absence of the former and with extreme care in the latter, as in the series reported by Bussenius, some infiltration may occur. Very marked systemic reactions occur in some part of the course of injections, but there is a reasonable suspicion that this may be caused by the apparent variation in strength of the preparation. It is possible that, if this uncertainty is overcome, immunity against the products of the microbe may be reached without undue reaction.

3. The immediate effects of the preparation upon existing lesions of the lung, larynx, bladder and middle ear are too indefinite to admit of any certain opinion being formed concerning them. In lupus, in various suppurating tracts, and in one noticeable case of tuberculosis of the uterus and its appendages, the remedy seemed to be of value; but whether of greater worth than the old tuberculin can only be determined by longer observations.

4. Koch's experiments upon guinea-pigs apparently established the fact that in them an immunity against both the bacteria and their products could be obtained, and, inasmuch as several patients after completing the course of injections stipulated by Koch received large doses of the old tuberculin without reaction, it would seem as if an immunity against the products of the bacilli could be produced in man. Whether such individuals possess also an immunity against the bacteria themselves, and therefore are protected

against reinfection, must be settled by observations extending over a longer period of time. The observation of Baudach in this connection is pertinent: "The question of the production of immunity is unsettled. If there is none produced, then the only point of difference between tuberculin R and the old tuberculin is the greater toxicity of the former." The class of cases in which the use of the remedy is justifiable is naturally very limited.

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December 9, 1897.

Appendix Vermiformis with Pin in Situ, Producing Interstitial Appendicitis.

JUDSON DALAND, M. D.

This appendix was removed from a male adult by Dr. H. J. Stubbs, of Wilmington, Del., to whom I am indebted for the opportunity of presenting the specimen to the Society.

Upon examination an ordinary pin was found occupying the lumen of the appendix, and, although causing no perforation, there were evidences of chronic appendicitis, as shown by the very considerable thickening of the walls, partial encapsulation and the presence of adhesions. For a number of months the patient had complained of pain in the appendiceal region, and presented the typical symptoms of acute appendicitis for ten days. The most marked symptoms were tenderness in this region with rigidity of the right rectus muscle. Twenty-four hours afterwards, the operation was performed and eight fluid ounces of pus removed, after which the appendix was ligated and removed. The temperature at this time was 103° F. The patient made a rapid and uneventful recovery and has continued in perfect health for more than two years.

The pin had become almost black in color during the time it had remained in the appendix. The patient had no idea how the pin gained entrance to the intestinal tract.

DISCUSSSION.

DR. RIESMANN recalled a case reported by Dr. Lloyd of a pin in a healthy appendix. Dr. Abbott stated that Dr. Welch had in his enormous experience in necropsies encountered very few instances of foreign bodies in appendices.

December 9, 1897.

Pneumothorax with Discovered Perforation.

J. M. ANDERS, M. D., and JOSEPH McFARLAND, M. D.

The patient was a man of thirty-five years, a compositor. His family history was negative. He had lived in good health until his present trouble began—about eighteen months ago. The first symptom was hemoptysis, which did not recur, and he was in good health afterward for one year, when gastric symptoms developed; pains passing toward the heart, nausea and infrequently vomiting. These symptoms have grown worse and he has also suffered from vertigo and headache. About six months ago he began to cough and a little later expectoration appeared; the sputum was at first mucoid, then muco-purulent. Emaciation and debility were constant and progressive. On admission to the hospital, November 4, the patient was very weak, pale and extremely emaciated. The gastric symptoms were then in abeyance. The urine was normal, the sputum contained tubercle bacilli, the blood showed a chloranæmia without leucocytosis. There were the physical signs of a cavity in both apices, small in the left and larger in the right. There were signs of consolidation on the left side below the cavity and on the right side there were feeble bronchial breathing and a semi-tympanitic percussion note. On November 11, the patient developed pain suddenly in the antero-lateral portion of the right side of the chest; urgent dyspnœa and some cyanosis appeared promptly, with a sense of impending suffocation. The pulse became rapid, small and irregular, the surface was cold, the temperature dropped to subnormal and remained there until death. The physical signs were typical of pure pneumothorax without the physical signs of effusion. The pathognomic coin-sound was readily obtained.

The autopsy was performed two hours after death. The body were much emaciated, the signs of physical suffering were marked.

The right lung was much collapsed. In the pleural cavity there were about 500 c.c. of a dark yellowish fluid containing some floating flakes of fibrin. Both lungs were adherent at the apices by old pleuritic adhesions, and both were the seat of pulmonary tuberculosis with excavations. As the patient had been known to have developed pneumothorax just before death, the rupture was at once carefully sought for, with the result that at the superior posterior aspect of the inferior lobe of the right lung, communicating with a cavity in the interior of the lung, there was a perforation of a diameter of 0.5 cm. in a thin layer of very necrotic material forming a layer about the size of an almond over a depression that entered the cavity. It is probable that the necrotic material consisted of the pleura and subjacent tissue in a condition of advanced tuberculous disintegration. The rounding of the edges of the depression probably took place after the rupture.

The heart was relaxed, its cavities nearly empty. There were no valvular or endocardial lesions. Death did not take place from asphyxia, but probably from asthenia. The abdominal organs, except the intestines, showed no important changes and the descriptions of their appearance will be omitted. Upon the serous surface of the intestines, here and there along the line of the lacteal and lymphatic vessels, there were strips of miliary tubercles. In the interior of the small intestine, especially in the ileum, there were occasional small ulcers.

The occurrence of pneumothorax is more common in tuberculosis of the lungs than in any other affection. It occurs, however, in simple abscess, in gangrene, in echinococcic affections, and in a few cases of traumatism.

Hermann Biggs has reported two cases in which pneumothorax has followed exploratory puncture of the chest with a hypodermic needle.

It also sometimes follows fracture of the ribs, and has been seen after gun-shot wounds.

Dr. Frey, Inaug. Diss., Zurich, 1888, studied 390 cases of tuberculosis of the lungs that occurred in five years in Eichhorst's clinic in Zurich, and gave the following statistics:

In 390 cases of pulmonary tuberculosis pneumothorax presented itself as a complication twenty times—or 5.1 per cent. Of these cases 18 were in men, and 2 in women. This makes the total percentage of cases in the male patients 6.6 per cent, in the female patients 1.6 per cent. Eighteen of the cases occurred on the right side, and only 2 on the left side. West, in a study of 101 cases of pulmonary tuberculosis, found pneumothorax in 5 per cent, but found that it occurred with equal frequency on both sides. Powell also observed the occurrence of pneumothorax in 5 per cent of his cases of pulmonary tuberculosis, and in the 17 cases that he reports we find the following figures: Left side, 10 cases; right side, 5 cases; double-sided, 1 case; indefinite, 1 case.

According to S. West, 90 per cent of all cases result from the rupture of tuberculous cavities. As Osler points out, pneumothorax usually occurs in adults, though at times it occurs in small children. The essential feature of the development of the condition is that there shall be an opening from the respiratory system into the pleural cavity without an opening of the chest wall.

While the other causes of the condition are of very rare occurrence, the figures already quoted have shown that the disease is of frequent occurrence in tuberculosis, occurring in 5 per cent of the cases. As seen in tuberculosis the lesion is almost invariably associated with cavity formation, so that it is usually a late complication of the diseases. The air escapes from the lung through laceration of the softened tuberculous tissue. In some cases the rupture of the softened tissue seems to occur without the intervention of any external cause, though often it can be referred to some unusual strain thrown upon the chest. Probably the most common causes are violent cough and sudden severe strains in muscular action, by which the air-pressure is elevated. For this reason the onset is almost invariably sudden, and characterized by severe pain in the side of the chest. Several dyspnoea, associated with the signs of threatened asphyxia usually attend the escape of air into the chest. The amount of air that can escape from the lung into the pleura must depend upon the amount of consolidation that has previously occurred, for it is obvious that in a consolidated organ it is impossible for enough collapse to occur to permit of the accumulation of much air in the pleural cavities. Eichhorst makes the generalization that pneumothorax is most apt to occur in cases of tuberculosis with rapid caseation and destruction of tissue. In the ulcerative forms with a chronic course, the thickening of the pleura and frequent inflammatory unions between the parietal and viceral pleuræ tend to protect the weak areas so that rupture is impossible. Only occasionally does the rupture take place at the apex. Ordinarily, it occurs at the lower border of the upper and upper border of the middle lobes. Most rarely it occurs at the upper border of the lower lobe. The most frequent point of occurrence is between the mamillary and axillary lines, on the level of the second or third intercostal spaces. It is usually said that when, in pneumothorax, the chest is opened the air escapes with an audible sound. Osler lays particular stress upon this, but in the case under consideration it was not noted, although it was expected and looked for.

Pneumothorax may be divided into the complete and incomplete forms. Of these the former is very rarely met. In it the lung is completely collapsed and compressed upon the vertebræ against which it lies. A third form is the circumscribed or sacculated. This form is seen in the cases in which the inflammation of the pluræ has caused more or less wide-spread adhesions between the two layers of the pleuræ.

Ventilated pneumothorax is the name given to occasional rare cases in which during inspiration the air is free to enter the pleural cavities, while during expiration there is some obstruction to its exit, by the action of some valvular fragment of pleuritic membrane, fibrin, etc.

At times there may be as much as 2000 ccm. of air accumulated within the chest. In almost every case there is not only air in the chest, but also fluid. It seems to be a rare occurrence that the air that enters the chest is sterile, hence in almost all cases there are added to the mechanical difficulties suppuration and the sequelæ from the infection. On this account the simple pneumothorax, soon becomes a hydrothorax, and this sooner or later a pyothorax. The outcome of the whole process sometimes being the formation of fistulous communications with the exterior.

Courtois-Suffit, *La Semaine Médicale*, December 15, 1897, has seen a case of pleuritis in which the symptoms became serious and paracentesis was done. A purulent fluid was removed, and a couple of days afterward extensive infiltration of the subcutaneous tissue with pus and gas was observed. After death, gas-forming bacteria were found in the pus. The case was probably one in which the gas produced by the bacteria transformed the empyema into a pneumothorax.

DISCUSSION.

DR. PACKARD.—I have observed a case in which an exploratory puncture of the chest was followed by sudden pneumothorax with profound shock, and early death. At the autopsy it was seen that the needle had perforated an emphysematous bleb of the lung. Thoracic puncture is certainly not entirely free from risk, especially in emphysematous subjects.

DR. STENGEL.—I have recently seen a thoracic effusion tapped in which, shortly after the operation the area became tympanitic, with amphoric physical signs; the area was localized in extent and the signs disappeared in a few days. I believe it was an aseptic pneumothorax produced in some way by the paracentesis. I believe also that the simple hypodermic needle may do damage, though I am certain that I have several times punctured the lung without bad results. I have seen death from perforation of the stomach and liver follow exploratory puncture, and would urge that there is some danger in these procedures.

DR. HUGHES.—I have had and seen the same experiences, which are, I believe, usually due to faulty instruments, and are usually harmless, except in emphysematious subjects.

DR. MCFARLAND.—In the cases which recover by absorption of the air what becomes of the nitrogen? In experimental work injections of air cause pneumothorax or emphysema, all of which disappears in the course of a short time.

December 23, 1897.

Congenital Inguinal Hernia.

DAVID RIESMAN, M. D.

The child, whose case I am able to report through the kindness of Dr. B. C. Hirst, was born, without mishap, after an induced labor, at the Maternity of the University Hospital, on December 3, 1897. On the fourth day after birth a swelling appeared on the right side of the scrotum, which on examination was found to be due to the existence of a complete right inguinoscrotal hernia, which was easily reduced and readily retained in the abdomen by means of a pad of cotton. On the third day the child had already presented slight fever; this continued to increase, without any other symptoms being added save progressive loss in weight. On the eighth day after birth death took place.

At the autopsy all the organs appeared normal except the suprarenal capsules, which were the seat of hemorrhagic infiltration. Cultures from the spleen and the adrenal bodies revealed the presence of staphylococci. I shall take the opportunity, on a future occasion, of reporting more fully on this feature of the case.

The point upon which I wish to dwell now, and which is illustrated so clearly in the body of the child, is the cause of the hernia. We observe here, on looking down into the pelvis, that the right inguinal ring is widely patulous, and can see the vas deferens passing down into open inguinal canal along the inner angle. The opening is fully 1.8 cm. in diameter, and its anterior inner edge is semi-lunar in shape and rather sharp. On the other side the inguinal ring is completely closed, and but for the vas deferens, it would be impossible to locate it accurately. Both testes are in the scrotum.

The child evidently had an open inguinal canal along which passed a loop of bowel, constituting the *hernia congenita* of Haller, which is the most frequent form of congenital hernia. The hernia in these cases passes down without any special sac of its own into the open funicular process of the peritoneum, entering in the scrotum that part which would normally constitute the tunica vaginalis testis. It thus comes to lie in close relation with the testicle, being in front of it, and being separated from it by only a single layer of peritoneum.

Congenital herniæ are quite frequent, as is shown by the statistics of the London Truss Society (quoted by W. B. Coley, *Medical News*, December 18, 1897) in which record is made of 21,000 cases of hernia, 3433 having been in children under the age of one year.

December 23, 1897.

Gangrenous Carcinoma of the Stomach.

MAURICE OSTHEIMER, M. D.

The patient, a woman of 54 years, very cachectic in appearance, was admitted to St. Joseph's Hospital October 25, suffering from procidentia uteri. Dr. Montgomery replaced the uterus, placing silver wire sutures in the vagina. The patient had for some years past complained of pain on the left side and in the epigastrium, where no tumor could be felt. Up to one month before her death, she ate well and retained everything. She vomited food but a few times during her six weeks at the hospital. About the middle of November she had a very profuse hæmatemesis which, two weeks later, recurred. After the first hemorrhage she could eat but little, though she still wanted everything. After the second she lived on whisky and ice until death, two weeks later, from exhaustion.

At the autopsy the diaphragm, stomach and the left lobe of the liver were intimately adherent, and the pancreas and spleen were also connected with the stomach by fibrous bands. Under the left edge of the diaphragm was a large sieve-like perforation in the anterior wall of the stomach, the area was gray and gangrenous and broke down upon touch, leaving a jagged oval opening about two and one-half inches long. Just above this and connected with it was a perforation one-half an inch in diameter through the diaphragm into the left pleura. That these perforations occurred post-mortem seems evident from the fact that there were no signs of peritonitis or pleurisy. The stomach itself seems small and the pylorus is constricted, while the gastric wall, from a band-like contraction two inches above the pylorus down to the outlet, is also much constricted. Upon the inner aspect of the stomach the gray gangrenous mass extends but one-half an inch around the edge of the jagged perforation, while the rest of the wall is covered with ridges and nodes of various sizes, white or grayish-red, projecting as much as half an inch above the surface. The wall, even when its thickness is not increased, is quite hard.

The left lobe of the liver is firmly adherent to the edge of the perforation, and is gangrenous at its tip. The connective tissue leading to the spleen and pancreas contains many small nodules.

I have some sections of fragments of tissue, taken from her last hemorrhage, two weeks before death, and hardened in alcohol. They show a few masses of epithelial cells in nests, which, taken with her pains, increasing cachexia and hemorrhages, seems enough, as Reineboth says in the *Deutsches Archiv für Klinische Medecin*, Band 58, Heft 1, to warrant treating the case as one of cancer of the stomach, if it is not enough to warrant a definite diagnosis of the case.

Discussion.

Dr. Sailer.—Noteworthy points in this case are the situation of the tumor, the extreme necrosis of the neoplasmic tissues and the mild character of the symptoms. I do not think a positive diagnosis of gastric neoplasm could have been made in this case from the appearance of the epithelial cells found in the vomita, although a cell-nest arrangement was exhibited, but considered in connection with the symptoms, it was quite conclusive.

December 23, 1897.

CARD SPECIMENS.

Gangrene of Lung.

D. RIESMAN, M. D.

The patient had had an acute pneumonia of the left lung; the sputum was bloody but not of the typical rusty appearance, nor had it the character of the sputum of pulmonary gangrene; the patient, however, exhaled a foul odor. Death occurred after eight days. In the left lung is a gangrenous cavity about 10 x 10 c. m.; the left pleura is covered with a fibrinous exudate; the fluid in the cavity presents no special microscopic characteristics.

Dr. Riesman also presented a lung from a case of advanced phthisis, preserved according to the method of Melkinow-Raswedenkow, in which the natural appearances of the fresh organ were well retained.

November 11, 1897.

Tumor of the Infundibulum.

DAVID RIESMAN, M. D.

Springing from the posterior aspect of the infundibulum and attached to the tuber cinereum is a small, pea-sized tumor, of a yellow color and firm consistency.

DISCUSSION.

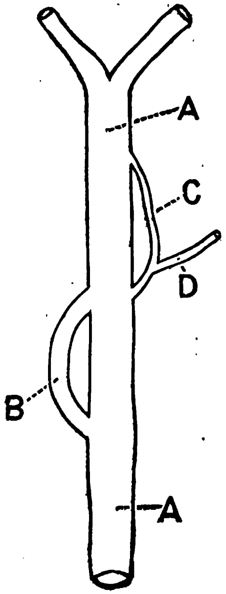
DR. H. W. CATTELL considered the neoplasm to be a fibrolipoma. In rare instances, however, teratoid growths were located in this space.

Anomaly of the Basilar Artery.

DAVID RIESMAN, M. D.

The artery is unusually long, extending from below the medulla to above the pons. It gives off from each side a short branch, which soon re-enters the vessel. (See sketch.) The left posterior cerebral artery gives off almost from the same part of its course two branches, which run parallel for a short distance, and are separated by a narrow slit, through which passes the third cranial nerve.

November 11, 1897.



Anomaly of basilary artery; a, a main trunk, b, c, d anomalous branches.

Slides Showing Basophile Cells.

MAURICE OSTHEIMER, M. D.

The first slide, a section of a tumor sent over to the laboratory of St. Joseph's Hospital, marked "from the cervix," is a non-malignant adenoma showing glandular substance with numerous basophile cells. These cells are stained purple with thionin.

The second slide is from a breast amputated at St. Joseph's Hospital for cancer. The nests of cancer cells are abundant, and there are many reddish basophile cells, stained by thionin.

These cells are quite numerous, irregular in outline, with faintly staining nuclei, and many dark basophilic granules.

December 23, 1897.

Proceedings
of the
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TABLE OF CONTENTS.

HUGHES, Specimens from two cases of Weil's Disease.—STENGEL, Cirrhosis of the Liver with Jaundice. Obstruction of Common Bile Duct and Suppurative Cholangitis.—McFARLAND, A Case of Symmetrical Gangrene of all Four Extremities Without Discoverable Cause.—WILLIAMS, Chorio-Epithelioma.—RIESMAN, A Case of Carcinoma of the Rectum, with Miliary Carcinosis of the Peritoneum.—WILLIAMS, Malignant Adenoma of the Intestine.—HAND, Tuberculosis of the Myocardium.—EDSALL and SAILER, Peculiar Bodies Found in the Central Nervous System.—KEEN and SPILLER, Two cases of Wrist-Drop from Injury of the Musculaspiral Nerve from Fracture of the Humerus.—HAMILL, Aneurysm of the Arch of the Aorta.—FISHER, Syphiloma of the Liver.—DUER and BLACKBURN, Colloid Cyst of the Ovary, Involving the Splenic Capsule.—KELLY, Multiple Sarcomata.—SAILER, Renal Cirrhosis.—STEELE, Ecchinococcus Cyst of the Liver.

Specimens from Two Cases of Weil's Disease.

W. E. HUGHES, M. D.

Case I.—The man was admitted to the Philadelphia Hospital November 23, 1897. On admission his chief complaints were restlessness, back-ache, chills and fever, jaundice and haematuria. The family and personal history was negative. Of late years he had lived in a malarial locality in Virginia till within two months, when he had removed to Baltimore, Md. He came to this city a week before his admission to the hospital. For some weeks back he had been out of work, and latterly, as a consequence, had been unable to obtain sufficient food. His clothing was scarcely enough to protect him, and his lodging places of the lowest order. In July, 1897, he had what was apparently an intermittent fever, the fever recurring at a regular hour for several days, being preceded by chilly sensations and followed by some sweating. Quinine was freely used, and its use was followed by a cessation of the paroxysms. In August he had another febrile attack, which was diagnosticated influenza. In neither of these attacks was there any jaundice or apparent abnormality of the urine. Since the July attack he had never become quite well and strong. About November 12 he caught a heavy cold, which grew rapidly and steadily worse. He became weak, nervous, restless, and complained of general aching, with especially severe pain in the back and lower limbs. He was chilly and hot by turns. On November 21 for the first time he began to pass urine of the color of blood, and he noticed that his skin was slightly yellowed. His appetite from the first had been poor; by this time he had begun to vomit.

On admission he was found to be a young man, slender, poorly nourished, the skin and mucous membranes uniformly yellow, his face thin, the cheeks sunken, the ears and lips blanched. The pupils were normal, the tongue was pale and covered with a whitish fur. Physical examination of the lungs showed nothing abnormal; over the heart, heard best at the base, was a soft blowing systolic murmur, probably haemic. The pulse was strong, rapid and fairly regular. The liver dullness was not increased, the spleen was enlarged, its tip being just palpable below the margin of the ribs. Temperature, 102°. A careful and exhaustive search was made in the blood for malarial organisms, but none were found. On the following day his temperature had fallen to 98.2°, the pulse was rapid, weak and irregular, and his general condition had grown worse. The jaundice had appreciably deepened. He had been vomiting bile-stained liquids, and the feces too contained bile. Several examinations of the blood for malarial organisms were made with negative results. In 24 hours 40 fl. oz. urine were passed; this was dark red, but the color was found to be due not to blood cells, but to haemoglobin; sp. gr. 1018, acid; there was a trace of albumin, but no tube casts. He died 36 hours after admission, with no notable change in symptoms.

Post-mortem examination by Dr. Riesman: The intestines were distended with gas; there was no fluid in the abdomen. The pericardium contained a small amount of bile-stained fluid. The heart was enlarged and flabby, the right auricle contained chicken-fat clots; there were clots and fluid blood in the right ventricle, and a clot in the left auricle. The valves were normal. The aorta was small compared with the size of the heart, and showed sclerosis in patches. The wall of the left ventricle was 16 mm., right ventricle 5 mm., in thickness. The lungs presented a few adhesions at the apices, were oedematous, and exuded a frothy, bile-stained fluid. The spleen, 17 by 11 by 3½ cm., contained a large hemorrhagic infarct; on section numerous white patches were seen, giving it a marbled appearance. Microscopic examination of pulp showed no malarial organisms. The adrenal bodies showed advanced fatty degeneration of the cortex, almost no cirrhosis. The kidneys were enlarged and firm, the capsules adherent, the cortices not diminished, the pyramids a brownish color; on pressure, a thin bloody fluid exuded. The bladder contained 3 fl. oz. of bloody urine. The liver was not enlarged; it measured 26 cm. long, the right lobe 22 cm., the left 17 cm. wide, 7 cm. thick. The color was fairly normal, in places bile-stained; the edges were sharp. The gall bladder projected 3 cm. below the margin of the liver; it was impossible to force bile through the common duct, despite which fact a probe could be easily passed from the duodenum to the gall bladder; the hepatic ducts were patulous and empty. The gall bladder was distended with bile too thick to pass through the ducts, resembling blackberry jam in appearance, but not gritty to the feel. The upper part of the small intestine was intensely bile-stained. On section, the liver was pale, marked by lighter areas, and its consistency was increased. Microscopic examination showed simply slight granular degeneration of the liver cells, with some round-celled infiltration of the capsule of Glisson. The bile contained no cholesterine, but there were peculiar crystalline masses of a cylindrical shape and of a red color. The bone marrow was reddish and rather firm.

Case II.—A man, *aet* 48 years, was admitted to the Presbyterian Hospital, February 16, 1897, under the care of Dr. De Forest Willard. For 24 hours he had been unable to pass urine. Some weeks previously there had been a similar condition, which had been relieved by catheterization. Two years ago he had gonorrhoea, since which time there has been a fairly constant urethral discharge, especially in the morning. There had been no difficulty in the passage of urine until two months ago, when the symptoms of a stricture became manifest. In the hospital a catheter was passed into the bladder with much difficulty, and that viscus emptied. Urinary infiltration of the scrotum, penis and lower abdominal parietes, which was present on admission, grew progressively worse, and for its relief, on the 18th, the bladder was opened through the perineum and numerous incisions made in the inflamed tissues. Following the operation he was much shocked, and through the night his bowels moved several times and he vomited frequently. The next morning there was a little jaundice present. This deepened rapidly, till by nightfall he was amost of a mahogany color. Through this day and the night the moderate amount of gastro-intestinal disturbance continued. He died early the next morning in collapse. At no time were there any cerebral symptoms. The urine was fair in quantity, sp. gr. 1020; it contained a small amount of albumin and many leucocytes, but no tube casts. The temperature ran about 100°, twice running up to 102°, once falling to normal. The blood examination was negative.

Post-mortem examination: The skin and all other tissues and fluids were deeply bile-stained. The heart was contracted; the cavities were empty or contained a small quantity of semi-fluid blood. The muscle was of natural appearance and thickness, the valves all normal. There were a few adhesions in both pleural cavities; at the apices of both lungs, a few small fibrous nodules and scars. In the small intestine some of the Peyer's patches toward the lower end of the ilium were a little swollen and had a shaven-beard appearance; there was no ulceration nor any apparent congestion; the mucous membrane elsewhere was normal. The lower bowel contained considerable light-colored fecal matter. The spleen was of normal size, firm, and of dark color. The kidneys were enlarged, the capsules adherent, the pelvis dilated. On section, the cortices were somewhat swollen and mottled, on pressure much blood exuded. The ureters were dilated and thickened, the bladder firmly contracted, its wall thick.

The liver, 1990 Grms., was much congested, the cut surface exuding blood freely, nothing else abnormal in its appearance. The gall bladder was moderately distended with fluid bile, all the bile ducts were patulous, their mucous membrane normal. Microscopic examination of liver showed pronounced cloudy swelling of cells. In most of the branches of the portal vein were rather firm blood clots. In the main trunk the clot reached just to the liver and here it was partly white and fibrinous; while this was the oldest part of the clot even here it was not laminated but had apparently been formed rapidly and rather recently. The clot was not adherent to the wall of the vessel, nor was there any diseased condition of the intima. It completely occluded the lumen.

While I have termed these cases of Weil's disease, I do so not because I am certain that this name exactly describes them, but rather that I am at a loss as to exactly where to place them. They are certainly cases of acute jaundice ending fatally, for which there is no mechanical explanation. From the results of the microscopic examination it appears that the jaundice is due to or at least associated with an acute inflammation or degeneration of the liver cells. Unfortunately no bacteriological examination could be made of either case, but the second is almost necessarily septic, while the first is more in doubt. In the first case the method of production of the jaundice is quite clear. The hemoglobinaemia caused such an excessive overproduction of bile-coloring matter that this was enough not only to cause the inspissation of the bile in the gall bladder but also by its absorption to produce the jaundice. The reason for the dissolution of the hemoglobin must remain unknown At the first the case looked like one of hemoglobinuric malarial fever that it was not malarial is evident from the fact that careful examination of the blood before and spleenic pulp after death failed to show any organisms. In the second case the clotted blood in the portal veins probably had some connection with the jaundice.

January 13, 1898.

Cirrhosis of the Liver with Jaundice.

ALFRED STENGEL, M. D.

I wish to present this specimen on account of the peculiar clinical characters of the case. The patient, aged 30, was admitted to the alcoholic ward of the Philadelphia Hospital with symptoms of delirium tremens. He remained in this ward for several days, where he became more quiet, and was then transferred to my care in the medical wards. At this time he was mildly jaundiced, and the temperature, which had been elevated during his stay in the other ward, had fallen almost to the normal. Subsequently it became slightly elevated. The patient vomited continuously, so that it was impossible to feed him. He was ordinarily clear in his mind, but occasionally rambled, especially in the evening. The liver seemed enlarged and the lower edge could be felt. The spleen was not apparently enlarged. Clinical examination discovered no other facts of interest. The urine became more and more bile-stained but neither leucin nor tyrosin could be demonstrated. The jaundice increased progressively in severity, and the vomiting continued until the patient was utterly prostrated, when the emesis ceased. Finally the patient died of exhaustion. At the autopsy the important pathological lesion was that in the liver. This was described as cirrhotic, with a nodular surface, and very firm in consistency. It was slightly reduced in size, measuring 30 cm. in length; nearly 18 cm. in width (both right and left lobe), the thickness was 18 cm. The edges were sharp. The gall-bladder, which was enlarged, projected 3 cm. beyond the edge in the liver. On section through the organ the general hue was grayish yellow, but this was dotted over with red and dark yellow areas. The surface of section felt very smooth, and the outlines of the nodules were obliterated. Microscopically, the appearances were those of old cirrhosis of the liver, with recent round-cell infiltration, but I leave the more accurate description of the appearances to Dr. Riesman, who examined the specimens.

I show the liver because of the peculiar clinical nature: the malignant jaundice. While icterus is not unusual in atrophic cirrhosis of the liver, and in fact nearly every case presents a certain change of color that is partially due to icterus, intense jaundice, such as this, does not often occur without definite complications, such as gall stones or other disease of the liver substance. During life we considered this case as probably hypertrophic cirrhosis, with possibly partial obstruction of the duct by catarrhal inflammation. Whatever the nature of the case might prove to be, we felt reasonably certain that it would not prove one of ordinary cirrhosis. Even now I am disposed to think there were peculiar elements in this case that do not ordinarily occur, and would refer particularly to the cellular infiltration, evidently of recent origin, to which Dr. Riesman will refer.

DISCUSSION.

Dr. RIESMAN.—The liver is the seat of an old, well-marked perilobular cirrhosis, with an acute process superadded, characterized by intense round-cell infiltration of the fibrous tissue of the capsule of Glisson. In places the width of the perilobular tissue exceeds the diameter of the encircled lobule. The bile-ducts are numerous, but in many instances the groups of epithelial cells in the capsule of Glisson appear to be hepatic cells that have become constricted off. The liver cells are in a state of cloudy swelling. There is marked biliary pigmentation, and in places bile-ducts are found that are filled with large clumps of dark-yellow pigment.

Through the kindness of the resident physician I was enabled to see this case during life. As acute yellow atrophy had been one of the conditions suspected, I was asked to make a culture from the blood procured through cutaneous puncture. I found staphylococci, but do not recall whether it was the staphylococcus albus or æureus. If such an examination can be relied upon, it may throw light on the case and explain the acute round-cell infiltration, so evident in the liver, as well as some of the clinical symptoms.

Dr. Eshner recalled (à propos) of toxæmia in hepatic disease, a case reported by him to the society years ago, in which death occurred with profound symptoms of intoxication eight days after the establishment of biliary obstruction.

Dr. Hughes.—Simple jaundice can scarcely be the sole cause of the toxæmia. Might it not often be renal, especially the more chronic cases? In the very acute cases, and in such as present marked intoxication, I believe that the most likely causes are bacterial.

Dr. Stengel.—Moderate jaundice, as I stated in my remarks regarding the specimens, may be occasionally an accompaniment of atrophic cirrhosis of the liver. When excessive jaundice occurs, we naturally look for additional causes. Eichhorst has recently described an instance of chronic atrophic cirrhosis in which acute interstitial hepatitis in the form of abundant cellular infiltration supervened and led to a rapidly fatal termination. Dr. Riesman's microscopic examination seems to show similar conditions here, though it is not apparent that the recent infiltration was the cause of the jaundice in this case. The classification of cirrhosis of the liver is not easy, and the frequent employment of the terms atrophic and hypertrophic as indicating merely large or small bulk of the liver without regard to the pathology or cause of the condition, is unfortunate. The classification which I have found the most satisfactory is that of Senator. He divides the disease into three groups: (1) Laennec's portal atrophic cirrhosis, of which there are cases (*a*) with hypertrophy, and (*b*) with jaundice. The jaundice may be due to complicated gastro-intestinal catarrh or to processes in the liver itself. (2) Biliary cirrhosis with subsequent atrophy, such as are due to long continued obstruction of gall ducts, with or without splenic enlargement. (3) Hanot's hypertrophic cirrhosis with jaundice. The termination of some of these cases in sudden death, perhaps in coma, is suggestive of toxemia, and in some cases distinct toxic conditions have been established, though the exact nature of the poisonous substances remains to be determined. I have not thought, however, that the condition could be termed uræmia. It resembles uræmia in a general way, as do other toxemias, but further than this our knowledge would scarcely warrant us in asserting a relationship.

January 13, 1898.

Obstruction of the Common Bile Duct and Suppurative Cholangitis.

ALFRED STENGEL, M.D.

The patient was a white woman, aged 76, who died in November, 1897. On admission to the hospital she complained of nausea, vomiting and pain in the abdomen. The family history and previous personal history were negative. Eighteen months ago she was seized with an attack of vomiting. This was followed by a chill lasting fifteen minutes. Fever and severe pain in the right hypochondrium ensued. The entire abdomen was tender. The skin became yellow three days after the attack began. During this attack she was sent to the German Hospital, where she remained five weeks, when she was sent to the Philadelphia Hospital. Ten days after admission she had a chill, followed in a week by two more. In three weeks she became better and left the hospital. She had cystitis while she was in the hospital, and also hemorrhoids.

In October, 1897, she was admitted again, with attacks similar to the former—fever and jaundice. The urine at this time showed a few pus cells and a trace of albumin, but no casts. In November she had an attack of vomiting, followed by a severe chill. At this time she had severe pain in the right hypochondrium, high fever, and rapidly became jaundiced. The heart and lungs were normal. She had a large reducible inguinal hernia on the left side. The abdomen was tympanitic and tender on palpation. There was slight induration over and some enlargement of the gall bladder The liver extended from the sixth interspace to one-half inch below the ribs in the right midsternal line. Operation was suggested to the patient, who would give no decided answer. She became comatose, the skin deeply jaundiced, and soon died.

Post-mortem Notes.—There was peritonitis involving the entire intestines. The descending colon formed the hernia and was necrotic. The gall bladder extended three inches below the liver. The lungs showed at both apices phthisical areas which had become calcified. The heart was normal; the membranes there, as elsewhere, stained with bile. The kidneys showed interstitial nephritis. The bladder showed cystitis; it contained a few ounces of purulent urine. In the duodenum was a pouch 1 ½ inches deep below the opening of the common duct. The pancreas showed three or four small abscesses in the tail, the head was adherent to the common duct and duodenum. The liver is of a dark color, smooth on section. Minute abscesses are seen associated with the peripheral ducts. All the ducts are dilated and thickened. The hepatic duct admits the large finger easily. The gall bladder is distended with liquid bile; no stones are present. In the cystic duct are two small stones. In the common duct near the duodenum are two large stones which completely block the duct.

January 13, 1898.

A Case of Symmetrical Gangrene of all Four Extremities Without Discoverable Cause.

JOSEPH McFARLAND, M. D.

The patient, a man aged about 40 years, single, an American, employed in the Adams Express Company, came under observation with no other history of ill health than that he had once, not very long ago, been treated in the University Hospital for "heart disease."

The patient was first seen at his lodgings by Resident Dr. McKenna, of the Medico-Chirugical College Hospital, on December 25, at 5 p. m. The patient was at this time in bed complaining of weakness and prostration. He was well bundled up in bedclothes but as soon as the doctor touched his hands he was astonished to feel how cold they were. Some blisters were observed on the fingers, and inquiry failed to bring to light any cause for them. The man had been well and had been at work delivering Christmas poultry, etc. Knowing that it sometimes happens that poultry is kept in cold-storage and preserved with solutions of lead, the doctor fancied that the skin might have been affected with lead from the poultry. Almost as soon as the doctor called his attention to the coldness of the hands, the patient began to complain of extremely cold sensations in both hands and feet. The pulse at this time was very small and fluttering.

At 7 p. m., December 26, the patient was admitted to the hospital. He was almost pulseless and the doctor counted 160 beats per minute. Whisky was administered and the condition improved somewhat. Later on the pulse could not be counted.

One ankle was complained of and the man remarked that he supposed he had sprained it. The member was examined but while both feet were swollen, the ankle was not sensitive and seemed all right. The feet were very cold and rather bluish. The hands were dressed again and some of the blebs opened. A yellowish serum exuded. The hands were still very cold and bluish.

Shortly after admission to the hospital the patient became restless and delirious, and as the night wore on, very violently so. In the delirium he tore the dressings from the hands, and it was then observed that one of the finger-nails and some of the epidermis remained adherent to them. After this the patient became quiet, turned over on the side and seemed to go to sleep. Three hours afterward, the doctor inquiring about him learned that he was still quiet, and investigated to see his condition. To the suprise of the attendant who had listened to his quiet breathing a short time before, the man was dead. The short time that the man had been in the hospital had passed without securing from him any history of himself or his illness beyond what is given. He was not examined physically as he was by mistake admitted to the surgical ward of the hospital as a case of "poisoned hands." The autopsy was held eight hours after death.

Autopsy.—Body of a fairly well-developed man of middle size, about forty years of age. The attention of everyone was at once directed to a peculiar condition of the hands and feet of the cadaver which were gangrenous. Both hands were symmetrically affected, the lesion embracing all of the fingers and the thumbs and extending up the dorsal surface to the third phalanges and up the palmer surface to the palms themselves. The diseased areas were of a purplish color or a dusky slate-color, gradually fading into the surrounding tissue without any attempt at the formation of a line of demarcation. The skin affected was slightly edematous, was here and there elevated in blebs which contained a rather turbid fluid, and was soft to the touch. Slight pinching was sufficient to detach the epidermis from the cutis, leaving a dark red shining surface beneath.

The right foot was involved to an extent including all of the toes, a small portion of the skin of the dorsum, and the plantar surface as far as the hollow of the foot. The left foot was less affected, the lesion being found most marked upon the great toe, and the tip of the second and third toes.

The ears, nose and other features were normal, *i. e.*, free of gangrene.

The body had been dead but a few hours, was fresh and well preserved and showed well-marked rigor mortis.

Thorax.—The *heart* was of enormous size and seemed to fill up the whole anterior part of the chest. It was relaxed and flabby and was empty except for a few small clots. The *pericardium* was normal in appearance and contained a normal relative amount of fluid. The enormous size of the heart suggested the existence of some marked valvular affection, but curiously enough the entire interior of the heart seemed to be free from disease. The walls of the organ were of normal relative thickness and seemed to be unchanged in structure. Minute examinations of the valves showed the presence upon their leaflets of a few minute scattered patches of atheromatous change that was quite insignificant and inconspicuous. The cause of the hypertrophy was not in the heart itself.

The right lung was intimately adherent to the thoracic wall by old adhesions, the left lung being free. Both lungs showed no pathological changes beyond an hypostatic edema.

Abdomen.—The *spleen* was intimately adherent to the surrounding tissues and was enlarged to the size of the hand. Its capsule was thickened and presented a fibroid patch of almost cartilaginous consistency about the size of a quarter-dollar, and somewhat stellate in form, suggestive of an old infarct. On section it was found to be purely capsular. The splenic substance was firm and fibrous. It was probably only changed by chronic congestion or local inflammatory conditions.

The *kidneys* were examined with particular interest as offering a possible hint as to the cause of the enlargement of the heart. Each organ contained an anemic infarct about the size of a small soup bean, and may also contain some smaller ones, as there are a few oval patches on the surface. Except for the infarcts the kidneys were about normal in size, perhaps a little thick, and appeared fairly normal. The capsule was of about the normal thickness. There was a marked congestion in irregularly distributed areas, the capsule could be removed without much difficulty, and was but slightly adherent. Except, therefore, for the infarcts and a probably small amount of arterio-sclerosis, the organs were apparently in good condition.

The *prostate gland* exhibited an interstitial hemorrhage on the left side, near which the veins were much enlarged.

The *liver* was enlarged, nutmeg in appearance, supposedly from increase in connective tissue. In all other respects the abdominal structures were normal.

The *brain and spinal cord* were removed with care and showed no macroscopic signs of disease.

Subsequent microscopic study of the organs, while it threw no light upon the etiology of the peculiar condition present, revealed as the most interesting tissue change, a tendency to deep congestion of all the organs with interstitial extravasations of blood, especially noticeable in the heart muscle and liver. In the latter the congestion occupied an intermediate area of the lobule, and was not so marked in the central and portal areas as in the capillary network between them.

No bacteriological examination by cultures was made, but the frequent hemorrhages into the tissues suggested a micro-organismal invasion and made us stain sections of all the organs with solutions and thionin and by Gram's method. It was impossible, however, to demonstrate the presence of any bacteria.

The chief interest of the case centres about its etiology. Whether or not gangrene is known to occur from *weakness of the general circulation* alone I am not able to answer, not having completed my review of the literature. Such a condition is, however, outside of my experience.

Atheroma, calcification of the middle coat of the vessels was absent.

Frost-bite is out of the question. The winter has not been severe and even if a man had slept out at night, he would not have frozen. This man lived a respectable life and has no history of exposure.

Ergotism is also out of the question, especially as the gangrene was so rapid and was of the moist form.

Embolism is to be thought of, especially as there were infarcts in both kidneys. There was, however, no cause of embolism apparent, the symmetrical nature of the condition opposes this view of the case, and both arterial trunks of the upper extremities were opened from the axilla into the hand without finding any arterial disease or obstruction.

Raynaud's Disease is a condition known to produce symmetrical gangrenous lesions, but they are of the dry variety, resulting from the shutting off of the blood supply. In looking over the literature I failed to find any reported case of Raynaud's disease in which four members were so extensively involved as in this case.

Nervous lesions, associated with decubitus, sometimes cause gangrene of the feet of a symmetrical type, but this man is known not to have had any such condition, and the macroscopical features of the brain and cord were normal.

Diabetes is an occasional cause of gangrene, usually of a local form, irregularly distributed in patches. A few cases in which there was a somewhat symmetrical distribution are on record, but nothing like this. The patient is known to have passed urine freely before death. His short sojourn in the hospital did not give an opportunity for an analysis or examination of the urine. After death, however, he was catherized and three of us examined for sugar with negative results. Nor was albumin present.

Albuminuria.—Symmetrical gangrene of the extremities in the course of renal disease have been reported by *Debove* (Bull. et mém. Soc. méd. de Hôp. de Paris, 1880, 2. s. xvii, 78–82), and *Roque* Thérap. contemp. Paris, 1882, ii, 689). This man, however, had no marked renal disease, the kidneys showing but slight arterio-sclerosis, and slight parenchymatous degeneration.

Saturnism.—Sainton (France méd., Paris, 1891, vol. xxviii., p. 221), reports a case of symmetrical gangrene of the extremities due to lead poisoning. The case recovered.

Traumatism, the most common cause of the moist form of gangrene is also out of the question.

We are left, therefore, in the dark as to the etiology of this case, and until future study shall throw more light upon it, compelled to regard it as *idiopathic.* Two similar cases, one of which was given a suspicion of ergotism, are on record and present marked similarity. These are reported by Thomas Camps (*Brit. Medico-Chirurgical Review*, July, 1855, p. 196), and Bernard Henry (*Phila. Med. Examiner*, vol. 12, p. 129).

DISCUSSION.

DR. SPILLER.—The short duration of the illness, the symmetrical envolvement of the distal portions of all four limbs, the numerous hemorrhages in the liver, and possibly the round-cell infiltration here, are suggestive of an acute infectious disease. The peripheral nerves and vessels might well repay microscopic examination, and cellular lesions might be found in the cervical and lumbar enlargements of the spinal cord if the method of Nissl were employed. Erb and Strümpell, among the first, advanced the idea that a neuron is more likely to show the first traces of degeneration in its distal portion. Neuritis does not commonly cause gangrene, but there are poisons (ergot, diabetes) which do produce such changes and also affect the nervous system.

DR. RIESMAN.—The round-cell infiltration is slight and would hardly indicate that infection was the cause of the gangrene. It seems to me, however, that the calibre of the aorta is quite small, and that this might account for the hypertrophy of the heart and also for the congestion found throughout the body. Is it possible that, in connection with the general congestion, produced by disproportionate smallness of the aorta and probably other parts of the vascular system, the moderate but constant exposure to cold entailed by his occupation in a cold-storage plant, could have led to frost-bite in the hands and feet?

Dr. White.—I recall a case which occurred in an inmate of the institution for the feeble-minded, a child, in whom following a period of coldness and pallor of the right foot and ankle, and the left foot, ankle, leg, and thigh, a painful gangrene set in in the areas mentioned, and rapidly went to a fatal termination. In this case there was thrombosis in the vessels, and disease in the nerves in the gangrenous areas, but not above the line of demarcation. The nervous organs have not yet been examined; no other lesions were present.

Dr. Eshner.—I agree that frost-bite has not been absolutely excluded, nor has Raynaud's disease. In any event we ought not to halt with the term "spontaneous gangrene."

Dr. McFarland agreed that the aorta was somewhat small, but did not believe that it was reduced enough to have produced the congestions, nor to have so abnormally predisposed to frost-bite.

January 27, 1898.

Chorio-Epithelioma—Deciduoma malignum.

H. L. WILLIAMS, M. D.

The first author to describe the development of malignant disease of the uterus subsequent to delivery was Chiari,[1] who published in 1877 an account of three cases of supposed primary carcinoma of the corpus uteri occurring in women, two of whom were under 23 years of age. All three cases developed and terminated fatally within six months after delivery. This important paper attracted little attention at the time, although recorded in the work by Rugi and Veit on "Carcinoma of the Uterus," published shortly after.

Tibaldi,[2] Guttenplan[3] and Meyer[4] a few years later reported malignant disease of the uterus developing soon after labor, in all of which the patients died, but it was not until the publication of an article by Sänger,[5] in 1889, under the title of "Zwei Aussergewönliche Fälle von Abortus," that general interest was aroused in this newly recognized disease.

Sänger remarked the presence of deciduous and placental elements as a characteristic, apart from all other uterine neoplasms, and designated the growth as a decidual-cell sarcoma.

In 1890 Pfeifer[6] reported a case of malignant hydatid mole, and in 1893 Sänger[7] drew still further attention to deciduous tumors of the uterus and contributed observations on the malignant character of their degenerations.

During the two following years very important contributions to our knowledge of malignant neoplasms developing after labor or abortion, were made by Gottschalk,[8] Schmorl,[9] Chiari, Rugi,[10] Pestalozza,[11] Fränkel,[12] Marchand,[13] Bacon,[14] Williams,[15] Paviot,[16] Jeaunel,[17] Lohlein,[18] Kossman,[19] Klein,[20] Hartmann et Toupet,[21] Nové-Josseraud et Jacroix,[22] Kottnitz,[23] Labensbaum,[24] Schants,[25] Tannen,[26] Kuppenheim,[27] Superno,[28] Resinelli,[29] Menge,[30] and Eden.[31] During the year 1896 still further contributions on this subject were furnished by Apfelstedt und Aschoff,[32] Lumberg und Mannheimer,[33] Morison,[34] Spencer,[35] Beach,[36] Durante,[37] Cazin,[38] Neumann,[39] Monod,[40] Van de Poll,[41] Kastrom,[42] Jones,[43] and Cock.[44]

The most recent valuable article on this subject is from C. Gebhard,[45] who reports three cases in detail (from one of which the specimens now presented before the Society are taken).

After going carefully over the literature at our command, we have been able to collect forty-five reasonably authenticated cases of the neoplasm under consideration.

Chorio-epithelioma, decidual cell sarcoma, syncytial carcinoma, or deciduoma malignum, by all of which names it has been described, may be defined as a malignant disease arising in the body of the uterus, either attendant upon or subsequent to pregnancy (originating, probably, at the placental site), and giving metastatic secondary growths in the lungs, lymphatic glands, brain, spleen and other tissues and organs, advancing with great rapidity, and leading invariably to a fatal termination.

The reason for the varied nomenclature of this disease is due to the dispute as to its histogenesis. It was first supposed by Sänger, on account of the close resemblance which the cells making up the greater part of the neoplasm and the metastatic deposits bear to decidual cells, to be a malignant degeneration of the decidua serotina. And since the decidua is formed by the hyperplastic connective tissue stroma of the endometrium, the name "decidual sarcoma," or "deciduoma malignum," seemed appropriate.

Subsequent investigations were conducted by C. Rugi,[46] Marchand[47] and others here referred to, and the theory maintained, which is now generally accepted, that the cells which were supposed to be decidual are in reality syncytial (maternal epithelium) and fetal ectodermic (Langhan's) cells, which have undergone malignant proliferation. There is, however, some likelihood that the growths reported under this title may have had histological variations and in some cases contained both proliferated Langhan's and syncytial cells, as claimed in one case reported; also that true deciduous cells were present in the cases reported originally by Sänger—as he still maintains,—his tumors resembling the type of sarcomata. But in the majority of cases the malignantly proliferated cells so closely resemble the syncytium in every particular, that it is impossible to deny their identity.

Since in this case the tumor has its origin in the epithelial covering of the chorion villi, and not from the decidual cells (as was supposed), the neoplasm is allied to the carcinomata rather than to the sarcomata; hence the name, now adopted, of chorio-epithelioma seems to be the better one.

As you well know, the ovum on becoming attached to some point of the uterine wall is enveloped by a fold of the endometrium, that part of the endometrium folded over the ovum being known as the "decidua reflexa," the portion between the ovum and the uterine wall the "decidua serotina," while the endometrium lining the remainder of the uterine cavity is known as the "decidua vera." The villi of the chorion, covered by their single layer of ectodermic cells (Langhan's cells), then become imbedded in the decidua serotina, which has upon its surface the single layer of cilliated cuboidal epithelium, with which the entire uterine cavity is lined. This superficial layer of epithelium is soon known as the "syncytium." We thus have two layers of cells, epithelial in origin, between the fetal vessels of the chorion villi and the connective tissue stroma of the endometrium, which latter become in fact the decidual cells of the serotina. In the normal development of the maternal placenta the decidual stroma becomes differentiated into two layers—the inner layer, in relation with the syncytium, firm and compact; the outer layer loose and ampullary, containing rich blood sinuses.

The syncytial layer soon expands, and, proliferating rapidly, forms a band of considerable width between the ampullary layer and the original superficial epithelium of the serotina. At about the sixth week the blood vessels of the ampullary layer rupture into this compact and proliferated syncytium, the tissue is entirely absorbed with the exception of the original band of syncytial cells, and the villi are left floating free in the maternal blood sinuses. As the fetal ectodermic cells are absorbed usually between the fourth and sixth week of embryonal life, the chorion villi are now covered by but a single layer of epithelial cells, the syncytium.

It is, then, according to the opinions which we accept, in this syncytial covering of the chorion villi, either during pregnancy or from villous remains after labor or abortion, that these malignant neoplasms have their origin.

The clinical history in connection with these cases is of special interest. All cases recorded have developed either during pregnancy or immediately after labor or abortion. Of the 45 cases which have been collected, none have occurred in primipera, but each has developed before the menapause. Seven cases have occurred in women under 25 years of age, and the majority in women under 30. Forty-five per cent of the cases have developed subsequent to hydatid mole.

The os is frequently open. The uterus is large, soft and boggy, and usually contains a soft tumorous mass that can be distinctly palpated. This growth is of placental consistency, soft, friable, and easily broken down and removed by the finger.

The uterine wall is early invaded by the malignant cells, and becomes soft and easily perforated.

Early metastasis and great malignancy are the special features of the disease. In the case reported by Williams, metastasis took place into the tissues of the vulva within two weeks of delivery, and before the development of any other symptom. Of the 45 cases reported, 60 per cent have given metastasis to and about the vagina. The next most common seat of metastatic deposit has been in the lung. In a number of cases recorded, metastatic deposits have also been mentioned in the brain, spleen, kidney, stomach, ovary, broad ligament, intestines, diaphragm, ribs, bladder, femur, liver or pancreas.

Hemorrhage of an intermittent character is a symptom usually, though not invariably, present.

In the majority of cases which have proved fatal, death has occurred in from 3 to 6 months.

A positive diagnosis of malignancy can be almost invariably made by microscopical examination of curetted material from within the uterus; hence the great importance, in view of the rapidly malignant character of the disease, of curetting all cases that show intermittent hemorrhages after labor.

The microscopic examination of these tumors and metastatic deposits show practically the same appearance, although somewhat varied in different cases. But in the majority, the greater part of the tissue will be seen to be composed of a fibrous reticulum, presenting marked alveolar and cavernous structure and containing cavities of various shapes and size, filled with fibrin threads and free blood and organized blood clots. These are often separated from one another by bands of epithelial tissue which bear a close resemblance to decidual cells. In this mass no trace is usually found of either blood vessels, lymphatic or glandular structure. Careful examination shows the presence of apparently two varieties of cells. Individual cells, varying from round or polygonal to spindle shape, according as they are free or closely packed together, constitute one part. Their size is very variable, and while some contain but a single nucleus, others may contain several, while still others may resemble the typical giant cell. The nuclei stain intensely, and in all points correspond exactly to epithelial cells.

In other areas of the tumor, bands and masses of protoplasm, more or less extensive, are found, which contain large, irregularly shaped nuclei that take the stain intensely. These nuclei are deposited in a homogeneous granular protoplasm containing vacuoles, without any differentiation or boundary between individual cells. Thin bands or ribbons of protoplasm are also found, containing nuclei extending almost entirely across them, but with no line of separation whatsoever between the cells. This structure resembles precisely the syncytial layer, which forms the superficial covering of the chorion villi.

Williams, of Johns Hopkins,[48] is of the opinion that what appear to be individual cells in various parts of the tumor—giant cells and cells containing several nuclei—are in reality simply cross sections of more or less extensive masses of syncytial protoplasm, containing free nuclei. If this view be accepted, all varieties of the malignant cells can be referred to a syncytial origin.

The fetal ectodermic cells, more or less goblet shaped, can be differentiated from the syncytial cells by the fact that the outline of their cell protoplasm can be clearly defined and that their nuclei stain more intensely. That these Langhan's cells have also been found in metastatic deposits has been reported by Gebhard[45] and others.

To summarize briefly, the most prominent histological features of the specimens here presented may be said to be: (1) A fibrous reticulum resembling organized blood, in the meshes of which are large spaces containing blood and fibrin, but no glands, blood vessels or lymphatics. (2) Bars, bands and islands of syncytial and Langhan's cells between the blood spaces, and in masses, throughout the growth. (3) Vacuoles in the syncytial protoplasm, and in some cases distinct chorion villi. (4) A small round cell infiltration in the fibrin and reticulum and also in the homogeneous protoplasm is also conspicuously present.

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A Case of Carcinoma of the Rectum, with Miliary Carcinosis of the Peritoneum.

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Of the divisions of the intestinal tract the rectum is one of the most frequent seats, if not the most frequent, of malignant disease, as is shown very clearly by statistics dealing with the subject. Leichtenstern[1], for example, collects from reported cases and autopsy-records of 616 rectal cancers to 121 of the colon, including the cecum and appendix, 16 of the ileum, and 17 of the jejunum and duodenum. The combined Viennese statistics of Maydl and Nothnagel show that there were held in Vienna, from 1870 to 1893 (twenty-four years), 41,838 autopsies—of these 3585, or 8.56 per cent, were on cancerous subjects. Of these latter cases 343 were intestinal cancers, distributed as follows: duodenum, 7; ileum, 10; colon, 164, and rectum, 162.

Regarding the frequency of rectal carcinoma in the two sexes, the majority of systematic writers state that it is most common in men; Kelsey[3], however, found a slight preponderance in women (57 women, 50 men). With respect to age, cancer of the rectum, like cancer of the other divisions of the digestive tract, occurs generally in advanced life, although several instances of the disease in young persons are recorded. Schöning[4] observed two cases in girls seventeen years old, Allingham[5] two in boys of seventeen and thirteen years, respectively, and Gross[6] a carcinoma of the anus in a man of twenty-two years.

A number of varieties of rectal cancers is distinguishable, surgical writers, as Albert[7], being in the habit of classifying them as squamous and cylindric epithelioma, scirrhus and colloid.

When we examine pathologic text-books on this point we find considerable confusion in the terminology. Hansemann[8], one of the latest writers on the subject of carcinoma, makes three divisions of those occurring in the large intestine (excluding, of course, the lowest part of the rectum in which squamous epithelioma has its seat), namely, (*a*) destructive adenoma, (*b*) cylindric carcinoma, and (*c*) medullary carcinoma. The last two may be either scirrhous or colloid. Ziegler[9] speaks of adenocarcinoma, colloid, simple, and scirrhous carcinoma. His simple carcinoma corresponds with Hansemann's medullary, but neither adjective is quite satisfactory. The first is an arbitrary term conveying no definite idea, the latter has been employed more frequently as a macroscopic than as a microscopic designation.

Adopting, however, for the present Ziegler's classification we find the adenocarcinoma to be the most frequent form. As my own case is of the simple (or medullary) variety, it may not be without interest to bring it before you, particularly since it is also somewhat unusual in other respects.

The patient was a man, 72 years of age, under the care of Dr. J. William White, to whom I wish to express my sincere thanks for the privilege of reporting the case. I am also indebted to Dr. Edson, resident physician in the University Hospital, for a transcript of the clinical notes. The man's history is meagre; he had hemorrhoids for a long time, but seemed to have been permanently cured of them ten years ago. For several years past he had occasional attacks of diarrhea, always yielding to treatment, until last August, since which time he had persistent looseness of the bowels. The discharges were chiefly mucoid in character, and gradually increased in frequency until, at the time of his admission to the University Hospital, he passed, under severe suffering, small quantities of blood-tinged mucus at very short intervals. At times a protrusion occurred from the rectum, the replacement of which gave him great pain.

On examination a tumor encircling the rectum was discovered, and a left inguinal colostomy was done. The operation gave but little relief; the patient lived about a month and then died in coma. Shortly before death ischiorectal abscesses developed and had to be opened. The urine, seventeen ounces in twenty-four hours, contained a trace of albumin and a few hyaline casts.

At the autopsy considerable fluid was found in the peritoneal cavity; the omentum was adherent in the region of the colostomy-wound, and also in the right iliac fossa. The lower margin of the omentum was thickened and fibrous and looked as if infiltrated with new growth. Adhesions existed in the region of the gall-bladder, and between the liver and the duodeno-pyloric junction.

The entire peritoneum of the intestines and the mesentery was the seat of a miliary carcinosis. The nodules were whitish and opaque and varied in size from that of a pin's head to that of a small pea. The peritoneum of the anterior abdominal wall was also affected, especially in the hypogastric region. In the flanks the nodules were very numerous. The peritoneum of the diaphragm felt rough, like seal-skin leather. The peritoneum was injected, especially about the larger nodules. There were no intestinal adhesions except those described and some rather firm ones in the right iliac fossa.

The caliber of the small intestines appeared about normal; the descending colon was small, but its wall was not thickened. The part below the artificial anus was contracted, thickened, and adherent to the wall of the bladder posteriorly. A finger introduced into the rectum through the anus met a firm stricture. The growth in the rectum began 3 cm. above the anus, and extended upward for a distance of 8.5 cm., surrounding the bowel completely. The lumen, though greatly lessened, was not entirely occluded. Laid open, the narrowest part of the lumen measured 2.1 cm., and was situated near the middle of the growth. The greatest thickness of the wall was 2.5 cm. The surface of the growth was generally smooth, in places congested, and in a few areas covered with patches of false membrane; on the left side it had a moth-eaten appearance, from the presence of small ulcers. Above the growth the bowel was not dilated, but the mucous membrane was thickened and pigmented. The kidneys were somewhat enlarged and firm; the capsule stripped with difficulty, and left a granular cortex. The latter was reduced in thickness and its color pale; the consistency of the organ was increased. The stomach showed a small whitish nodule just be-beneath the peritoneum on the anterior surface of the fundus.

The liver was of normal size, its edges rounded. Only one nodule was seen on the surface, but on section several small and one large one the size of a horse chestnut, were seen. The nodules were whitish, very sharply defined, and surrounded by an area of congestion. The lateral lobes of the prostate were enlarged.

Microscopic Examination. The rectal carcinoma.—The growth is a simple or medullary cancer—the alveoli are small and are filled with solid masses of epithelial cells, which are not seen very distinctly on account of the highly cellular character of the stroma. Nothing of the mucous membrane proper remains. The cancer-nests are for the most part rather long and narrow, and run either vertically or horizontally to the rectal surface. In places the stroma has undergone hyaline degeneration, and that serves to bring out the small cellular nests more completely. Numerous dense collections of small round cells (abscesses) are present in the section, especially about the capillary blood-vessels. Several large vessels filled with blood are seen, from which hemorrhage into the surrounding tissue has taken place. Their perivascular lymph-spaces are infiltrated with carcinoma; round-cell accumulations are also contiguous to these large vessels. In the deeper parts of the tumor the cancerous cell-masses grow larger and their relation to the blood-vessels—they are in the perivascular lymph-spaces—becomes more striking. The muscularis is enormously thickened, and to a large extent infiltrated with the new growth, especially the circular layer. About the epithelial nests there is a marked round-cell accumulation. The subserous layer shows much congestion of the vessels and cancerous involvement of the subperitoneal fat.

The carcinomatous nodules of the *liver* resemble the primary growth. They consist of fairly large, solid nests, surrounded by broad bands of dense fibrous tissue. In the largest nests an extensive necrosis has taken place, which in some is total, in others spares the peripheral cells. Although the affected areas refuse to stain, the outlines of the original cells can be dimly made out. The liver-cells contain granules of bile-pigment, and have a hazy, granular protoplasm. Round-cell infiltration is seen about many perilobular bile-ducts, and round cells, arranged generally in single file, have penetrated between the columns of liver-cells.

The *kidney* is the seat of an intense parenchymatous degeneration, which is more than cloudy swelling, and really partakes of the character of a necrosis. There is a slight hyperplasia of the connective tissue about the tubules and glomeruli, and an exudate into some of the capsular spaces. The *spleen* presents marked hemorrhagic infiltration, otherwise nothing abnormal.

Clinically, it is interesting to note the existence of diarrhea, really dysenteric in character, during the course of the disease. To this symptom of rectal carcinoma Nothnagel has called attention.

The immediate cause of the patient's death is a subject deserving a few words of discussion. In view of the renal changes it is probable that uremia played a part, but it is not unlikely that the acute carcinosis was also a factor. There is, of course, no proof for the assumption that the coma—which lasted thirty-six hours—was "carcinomatous" in character, by which is meant that it was peculiar and partook of a nature somewhat analogous to diabetic coma. Regarding the influence of the acute peritoneal carcinosis, however, it is not unreasonable to assume that the active proliferation of cells which resulted in the formation of countless nodules, was attended by the elaboration of abnormal metabolic products which entered the blood-stream and poisoned the system—perhaps the acute necrosis of the renal cells was in part a consequence of the action of such poisons. These phenomena, if real, would not be without analogy—miliary tuberculosis kills, as is well known, by systemic poisoning, which is generally attributed to the toxic substances derived from the tubercle-bacilli. I would not minimize these effects, but is it not probable that the feverish cell-proliferation giving rise to innumerable tubercles produces harmful metabolic substances or, perhaps, abstracts from the blood material needed for other purposes? Whichever of the latter views be the true one, the effect of the miliary tuberculosis, as of the miliary carcinosis, would be to alter the composition of the blood and thus to render life impossible.

I have omitted any reference to a possible exogenous poison in carcinoma, derived from the hypothetic parasite. Of course, if such a parasite exists, it may contribute its share to the toxic substances in the blood.

A few words more about the anatomic findings in the case. The peritoneum, as described, was studded with small, opaque, whitish nodules, resembling miliary tubercles, but a trifle larger. In the region of the diaphragm and the suspensory ligament of the liver the disease took a somewhat different form—instead of circumscribed nodules there was a general fibrous thickening, with roughening of the serous membrane which, macroscopically, suggested a fibrous peritonitis merely, and not malignant disease. The microscope, however, clearly demonstrates the cancerous nature of the induration.

The secondary deposits in the liver were deep-seated, few in number, and unattended by any enlargement of the organ. The hepatic involvement, evidently, was older than the peritoneal, and was no doubt the result of hematogenous (portal system) metastasis, while the latter was lymphatic in origin.

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January 27, 1898.

Malignant Adenoma of the Intestine.

H. L. WILLIAMS, M.D.

I am indebted to Mr. Phillips, a member of the graduating class of the University, and to Dr. Burns, of Scranton, for the privilege of showing the present specimen, which was given me for examination with the following facts:

The patient from whom the growth was removed was operated upon by Dr. Burns more than a year ago. For some time previous to the operation the man had noticed a small nodular mass in the left inguinal region. This increased in size, and finally caused almost complete intestinal obstruction.

The patient was then operated upon for a supposed carcinoma of the sigmoid flexure.

On opening the abdomen, a distinctly circumscribed mass in the continuity of the bowel was found, and the tumor which I here present was exsected. The patient recovered, has an artificial anus, and has had no recurrence.

The tumor consists of a circumscribed growth of the intestine two and a half (2½) inches in length. The diameter of the mass as a whole is one and three-quarters (1¾) inches. No ulceration or granulation appears on the external surface of the intestine, which is richly supplied with fat. The tumor has a hard, resistant feel resembling a solid growth. On section, the lumen of the gut presents a granular, worm-eaten appearance; the wall of the intestine is greatly thickened, and measures from ⅛ in. to ¼ in. in thickness. The calibre of the gut at its most restricted portion would scarcely permit the passage of a lead pencil. On microscopic examination the diseased tissue is seen to be made up almost entirely of the glandular structure typical of malignant adenoma. The submucosa has been entirely destroyed and the muscularis invaded, but the serous covering of the bowel has not been at all affected. Certain areas show a proliferation of the lining epithelium, a rupture of the glands, and the characteristic epithelial invasion of the stroma, giving us the typical picture of adeno-carcinoma.

According to Ziegler, a malignant adenomatous tumor may occur either in the intestine or stomach which shows histologically only the typical appearance of simple tubular adenoma. This variety destroys rapidly the various layers of the bowel, and gives metastasis to other organs of the body.

The glands in the present specimen, however, are of the atypical type that are seen in malignant adenoma of the uterus. The atypical form of these glands may best be described by comparing them to a solid mass of lumbracoides cut in cross section—i. e., glands so closely packed, irregular in outline, twisted and intertwined together, that the continuity of an individual gland cannot be traced throughout its entirety, but is immediately lost or runs into that of another. This complicated microscopic appearance is found in most parts, though not in all parts of the preparation.

The interstitial stroma is greatly reduced in extent, and in places almost entirely wanting, and the glands frequently come together and become agglutinated.

To summarize briefly, we consider the principal microscopic points of malignant adenoma to be as follows: (1) The atypical adenomatous glands resembling lumbracoid forms, as above described. (2) Absorption of the stroma and agglutination of glands, or loops of the same gland. (3) Proliferation of the lining epithelium of the glands. (4) Filling of the gland spaces, rupture of the lumen of the gland and proliferation of the epithelium into the surrounding stroma.

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Tuberculosis of the Myocardium.

From the Laboratory of Pathologic Histology, University of Pennsylvania.

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The literature of this subject has been reviewed recently by Labbé (*Rev. mens. des mal. de l'enfance*, June, 1896), and by Barié (*Sem. Med.*, 1896, Vol. iv, No. 50), the former reporting two, the latter one case. The first report on record is that of Townsend in 1832. Barié, in his paper, which is the latest, collects thirty-nine cases, including his own. The great majority of cases are secondary to tuberculosis elsewhere, but two reporters, Demme and Knopf, considered their cases to be primary.

Lancereaux has described the morbid anatomy, three different appearances being found. (1) The first is the large, gray tubercle, varying in size from a pea to a hen's egg, usually multiple, and resembling the syphilitic gumma, but differing from it in having no envelope of fibrous tissue, and in the presence of giant cells and tubercle bacilli. (2) The second and rarer form is miliary tuberculosis, many tubercles being scattered throughout the muscle, some being visible beneath the endocardium. (3) A very rare form, also, is the genuine tuberculous myocarditis, with sclerosis, areas of small round-celled infiltration with giant cells being found between the bands of fibrous tissue or the muscle fibres. I have to report two cases, one belonging to the first variety, the large tubercle, the other to the second, miliary tuberculosis.

With regard to the transmission, Saenger states that it is through the lymph-channels, from the mediastinal glands. Barié admits that this does occur in some cases, but that sometimes, especially in miliary tuberculosis, the transmission is via the blood-current. In both of my cases this was evidently the mode of infection. A third channel is by direct extension from the pericardium. The usual seat of the tubercles is in the ventricular walls; next in order, in the right auricle. Valentin found the tubercle-bacilli to be few in number and my experience has been the same.

Symptoms are often wanting, and when present are only those of a myocarditis, with nothing distinctive to indicate the form. The symptoms that have been observed are dyspnea, gallop-rhythm, tachycardia, bradycardia, arrhythmia, palpitation and cyanosis. Brosch (*Wien. Med. Presse*, 1896, No. xxxvii) has reported a case in which the process spread from the mediastinal glands to the pericardium, then to the myocardium, ultimately ulcerating through the endocardium and giving rise to Weil's symptom-complex. This case and the two herewith reported raise the total number of cases on record to forty-two.

Case I.—The first of my cases was observed in the wards of the Children's Hospital, while substituting for Dr. Stengel. The subject was 5½ years of age, a female; the family history was negative. As an infant she had malnutrition. Pertussis attacked her ten months and catarrahal pneumonia two months before admission. Following the latter illness the cough continued, emaciation was progressive, and there was diarrhea. The examination of the lungs revealed increased vocal fremitus over the right upper lobe, with small moist râles on both sides. There was much dyspnea. The abdomen was swollen and the superficial veins were dilated. Death occurred two weeks after admission, being preceded for two days by meningeal involvement, as evidenced by convulsive movements starting in the right side of the face, spreading to the right arm and leg and later becoming general. There was also rigidity of the neck-muscles.

Post-mortem examination, 3 hours after death.

Abdomen:—The liver was enlarged to three fingers' breadth below the costal border; the enlarged part being adherent to the parietal peritoneum; over the posterior and superior part of the right lobe the adhesions are well organized; gray tubercles were sprinkled over the surface. Weight of liver, 528 grams. Microscopic examination showed slight fatty degeneration with miliary tubercles. The spleen, which is here shown, is surrounded by adhesions and filled with pea-sized, yellow tubercles, each one being surrounded by a bright red margin of congestion; weight 98 grams; the shape is curious, there being, in addition to the regular structure, an extra lobe or keel-like projection on the inner surface. Microscopic examination of the tubercles and surrounding tissue confirmed the gross appearance. The kidneys were enlarged and pale, the capsules stripping easily; small tubercules were seated in cortices and medullæ. Weight of left, 57 grams; of right, 59 grams. Microscopic examination shows, in addition to the tubercles, a degeneration of the epithelium in the convoluted tubules. The omentum was markedly tuberculous, with a thick tuberculous band, 2½ cm. broad, along the free border. The mesenteric glands were enlarged and some were caseous. The intestines were normal, except for two follicular ulcers in the lower part of the ileum. The other organs were normal.

Thorax:—Pleural adhesions were quite general, and soft, except toward the apices where they were firm. The bronchial glands were very large, yellow and caseous. The right lung was filled with small hard tubercles, weight 153 grams. The left lung was consolidated in its entire extent except along the anterior and lower borders; in the upper lobe is seen a cavity the size of a horse-chestnut; on the bronchial mucous membranes were ulcers. Tubercles were situated on the pericardial layer of the left pleura. The pericardium was normal.

The heart weighed 52 grams; its external appearance was normal; the left ventricle was empty; other chambers were filled with currant jelly clots. On the left side of the septum ventriculorum, just beneath the endocardium, was a pearly tubercle, which, microscopically, consists of small round cells. The aggregation was undoubtedly of tuberculous origin, although the demonstration of bacilli failed, through an unfortunate accident.

Brain:—The meningitis which existed was miliary, the tubercles being of general distribution, but most numerous along each side of the great longitudinal fissure in the posterior frontal and parietal regions.

Case II.—This case was in the eye-ward of the Children's Hospital, in charge of Dr. B. Alexander Randall, through whose courtesy the specimens are presented. The patient was 5½ years of age, a male, colored, who was admitted in October, 1896, with a discharging fistula below the left eye. The bone was found to be diseased, and the discharge continued, giving rise to a most putrid odor. There was a history of convulsions before admission, but none were noticed in the hospital. The boy was stupid and somnolent, moaning during sleep. On June 8, sudden protrusion of the eyeballs occurred, the temperature rose to 105° and death ensued. Post-mortem examination twelve hours after death. Brain:—There was an extra dural caseous mass along the upper border of the left squamous bone, with thickening of the bone and the formation of spicules. There were pearly tubercles over the cerebellum, and a tyroma in the right temporal lobe. The brain substance was very soft. The right cerebrum was bathed in greenish pus. The cavernous sinus was filled with a putrid thrombus, and the torcular herophili contained a chicken-fat thrombus which extended into all the sinuses.

Thorax:—The right lung showed hypostatic congestion at base; there were many tubercles in the apex. The left lung was more extensively tuberculous, with a cavity in the upper lobe. The bronchial glands were enlarged. The heart, which I here exhibit, cnotained chicken-fat clots in all the chambers; at the apex in the wall of the right ventricle is a mass the size of a hickory nut, appearing yellowish-white under the pericardium. This mass is found to extend through the wall, and is visible on the inner side. Microscopic examination showed caseous degeneration and small round-celled infiltration, with giant-cells. A few tubercle-bacilli were found.

The intestines were the seat of extensive tuberculous ulceration, involving the ileum, colon and appendix, with the formation of tubercles on the peritoneal surface.

The liver, spleen and kidneys were not tuberculous.

January 27, 1898.

Peculiar Bodies Found in the Central Nervous System.

FROM THE PEPPER LABORATORY OF CLINICAL MEDICINE.

D. L. EDSALL, M. D., and JOSEPH SAILER, M. D.

The amylaceous bodies found in the central nervous system are fairly constant features in association with the changes occurring as a result of arterio-sclerosis. Other accumulations have been described by Rosin and various other writers, some of which are unquestionably artefacts, as those that appear occasionally in tissues that have been kept too long in alcohol; others appear to be pathological, although their nature has not been definitely determined.

We have recently observed peculiar concretions, that appeared to bear a certain resemblance in two cases of very different nature, and although they appear in some respects to belong to the usual types of degeneration, they do not respond to all the characteristic tests for any one.

The first case was typical tuberous gliosis. In addition to the nodules in the cortex, there were numerous small papillary elevations in the floors of the lateral ventricles. These were found to consist of dense masses of coarse irregularly arranged fibres, and in the meshes of some of these nodules, peculiar bodies were found. They may be described as rounded, irregular masses, sometimes as large as a millimeter in diameter, and often distinctly concentric in structure. They are not apparently connected with the blood vessels, and indeed, in those situations where they are found, the tissue is usually entirely without cells, and only slightly vascular. The vessels that are present are sometimes normal, sometimes exhibit a slight hyaline degeneration of the walls. In some of the nodules none of these bodies were found, and in a few of these, as well as in some of the sclerotic areas in the cortex, little groups of dilated capillaries were present. Examined in unstained sections (the tissue had been hardened in Müller's fluid) they appeared as ill defined, homogeneous masses, paler than the surrounding tissue. When the section was immersed in an iodin solution, they remained pale, but became more distinct by contrast with the partially colored surrounding tissue. With Delafield's hematoxylin they stained a deep blue; with Weigert's a deep brown; with Van Giesen's and Rosin's stains, a clear red; and although they failed to give the amyloid reaction with iodin and sulphuric acid, they stained a deep red with gentian violet, much better than is usually obtained with true amyloid substance after it has been imbedded in celloidin. Eosin was only used in connection with hematoxylin, and as the latter stained them deeply, we cannot say whether they have also a special affinity for the acid stains. Peculiar bodies have also been described in other cases of tuberous gliosis; thus Bruckner[1] notes the existence of large glassy round cells, between which were chalky deposits in the fibrous stroma extending inwards from the ependyma; and Pozzi,[2] in another case, found certain masses in the midst of the neuroglia, that he regards as colloid. In other cases they either did not exist or their presence was overlooked.

The second case was that of an infant eighteen months old, who when first seen by one of us had well-defined symptoms of tubercular meningitis, but the presence on the right side of a hemiphlegia at once attracted attention. The parents, who are not intelligent, did not know how long this condition had existed. At the autopsy, in addition to the meningitis, a large area of degeneration was found in the left internal capsule, involving the optic thalmus and the ventricular nuclei. In the midst of this area numerous irregularly round masses were found that gave the following color reactions: iodine either alone or combined with mineral acids fail to stain them, in fact as it did stain the surrounding tissue a faint yellow, they appear, as did those in the other case, to be clearer and more distinct than before its use. Delafield's hematoxylin stained them a faint bluish purple. When the section was immersed in a solution of gentian violet these bodies stained a bright red color. With thionin they stained a reddish violet. They did not stain with Van Giesen's, or with Rosin's, or with carmin. (The tissue had been hardened in alcohol.)

The nature of these bodies is difficult to determine. Dagonet[3] has figured something very similar in appearance and called it hyalin degeneration. That they belong to the group of degenerative processes that includes amyloid, hyalin, colloid and mucoid degeneration is, of course, obvious from the color reactions. These evidently do not represent distinct chemical compounds, but a group of substances that probably can pass one into the other; and von Recklinghausen has included them under the name of colloid degeneration and believes that they all originate from the protoplasm of the cell. The best distinctions that we have are apparently the reactions with the stains. Colloid substance stains orange with Van Giesen; hyalin substance a bright red; neither stain with iodine, which gives a deep brown color with amyloid substance. Mucin should give none of these reactions. Alzheimer[4] has recently discussed the subject of colloid degeneration of the brain, using the name according to the idea expressed by von Recklinghausen. He collects a number of cases of this condition, of which the most important, from our point of view, on account of the occurrence of small bodies not directly attached to the blood-vessels, are those of Arndt, Maier, Schüle, Liebmann and Holschewnikoff; but in all these cases the vessels were also extensively diseased. He also reports two cases of his own. The first had been diagnosed dementia paralytica, but at the autopsy extensive colloid degeneration of the vessels, with colloid masses in their immediate neighborhood, was discovered. These masses gave the following reaction: red with carmine, Van Giesen's stain and eosin, even if hematoxylin were used; dark blue by Weigert's fibrin method, although the older masses remained pale; these latter, however, gave a characteristic amyloid color with methyl violet and iodgreen, and stained brown with iodine, but did not give the amyloid reaction. The primary changes appeared to be invariably in the walls of the blood-vessels and to extend from them. The second had been diagnosed brain tumor. Colloid degeneration was found identical with that of the first case. Whilst in the ordinary acceptation of the term, these changes would not be called colloid, there are various reasons why they differ from those of our cases, of which the most important is the presence of vascular changes. Ziegler[5] describes a form of hyalin degeneration that occurs in scars, connective tissue tumors, and degenerated thyroid glands. It is similar to the form of hyalin degeneration occurring in the intima of the vessels and the endocardium of the heart. According to von Kahlden, this hyalin substance, as well as colloid material, stains with hematoxylin. In favor, however, of regarding the masses, particularly in the first case, as slightly modified amylaceous bodies, is their concentric structure, and their situation beneath the ependyma of the ventricle, where these bodies are usually collected in sclerotic conditions. It is not necessary to consider them as originating them from the blood-vessels. Redlich[6] has already called attention to the fact that they can exist independently of these, and suggests that they may be due to degeneration and conglomeration of the neuroglia cells. Stroebe[7] and Dagonet suggest, with more apparent reason, that they are products of the destruction of the myelin sheaths. Particularly in our second case, this explanation would be satisfactory, on account of the extensive degeneration of the medullated fibres and the absence of fat in the tissues. In conclusion, we are inclined to look upon them as degenerative products situated chemically somewhere between amyloid and hyalin substance, but not exactly identical with either.

REFERENCES: (1) Bruckner.—Archiv. f. Psychiatrie, vol. xii, No. 3, 1882. (2) Pozzi.—L'Encephale, p. 157. (3) Dagonet.—Système Nerveuse Centrale, Paris, 1897. (4) Alzheimer.—Archiv. f. Psychiatrie, vol. xxx, No. 1, 1898. (5) Ziegler. —Lehrbuch der Pathologie, 1895. (6) Redlich.—Jahrbuch f. Psychiatrie, vol. x, 1891. (7) Stroebe.—Ziegler's Beiträge, vol. xv, 1894.

DISCUSSION.

DR. SPILLER.—The amyloid bodies of the central nervous system are homogeneous, round, stain a light purple with Delafield's hematoxylin, and are especially numerous in the posterior columns of the spinal cord in persons advanced in life. They do not give all the reactions of amyloid substance. These bodies described by Drs. Edsall and Sailer are larger and more irregular in shape than the amyloid bodies. They may, perhaps, belong to the peculiar formations pictured in Dagonet's book, only the latter seem to have some connection with the vessels, and present more of the appearance of a mulberry than those shown us to-night. It seems to me exceedingly probable that the bodies described by Drs. Edsall and Sailer represent some form of parenchymatous degeneration, possibly hyaline in character, and as the amyloid bodies are supposed by some to be derived from the axis cylinders, these possibly may represent altered myelin.

January 27, 1898.

Two Cases of Wrist Drop from Injury of the Musculospiral Nerve from Fracture of the Humerus.

W. W. KEEN, M. D. and W. G. SPILLER, M. D.

Case I.—A boy fractured the right humerus just above the elbow. The paralysis of the extensor muscles of the hand was first noticed when the splint was removed, five weeks after the accident. Electricity was employed for six months without any benefit, and there was progressive atrophy of the extensor muscles of the forearm. The patient could still supinate the forearm, but could not extend the hand. The index and middle fingers, from the distal end of the proximal phalanges to the extremities, were anesthetic. An operation was performed and the nerve was found to be thickened, but not divided. A portion, 65 mm. long, was removed and examined microscopically.

The nerve fibres were found in a far better state of preservation than could have been expected, in view of the extensive paralysis which had lasted for nearly two years. In two or three nerve bundles, very decided changes were noted; the medullary sheaths were much less numerous than in normal nerves and, therefore, the hæmatoxylin of Weigert did not stain these fasciculi deeply, and the carmine stain showed some swelling of the axis cylinders. The epineurium, perineurium, and endoneurium were not notably thickened. The moderate swelling of certain axis cylinders indicated that the process was progressive, for this is an early stage of degeneration. Far more than half of the fibres appeared to be normal. It is evident, therefore, that complete paralysis may be the result of injury of a comparatively small number of fibres within a nerve, and this fact is one of considerable importance.

Case II.—The second case was a boy who had had wrist drop for a number of years from fracture of the humerus. At the time of operation he could not extend the hand at the wrist and had very imperfect extension of the fingers, this being limited to the second and third phalanges. The right forearm was much shrunken, the shoulder and elbow movements were not impaired, and the supinator longus muscle, in this case also, was intact. All the extensors of the forearm were completely paralyzed, and showed, in an examination by Dr. Bachrach, quantitative and qualitative changes in the electrical reactions. Dr. Keen found complete division of the musculospiral nerve. Both ends were much enlarged and these were removed, and the gap of 6 cm. between the two portions of the nerve was bridged over by catgut sutures.

The much thickened proximal end was examined microscopically. In transverse sections, from the upper portion, the nerve fibres were, in great part, arranged in bundles, but within the thickened epineurium many nerve fibres were found passing in all directions without any definite arrangement. The connective tissue of the nerve was greatly increased in amount. The longitudinal sections through the middle and lower portions of the swelling showed the nerve fibres almost without the normal arrangement in bundles. The fibres were spread all over the field; were separated from one another by connective tissue; some ran transversely, but most had a longitudinal course. It is probable that these were new fibres which had grown from old axis cylinders and had become wrapt, each in a medullary sheath. They did not stain quite as intensely with the Weigert hematoxylin stain as do ordinary nerve fibres. It is hardly possible that they were merely the old fibres, which had been pushed apart by the proliferating connective tissue of the endoneurium. The swelling was, therefore, a true amputation neuroma.

Within a short time after a nerve has been divided, several new axis cylinders grow from an older one at some little distance above the point of division; each then becomes surrounded by a medullary sheath and constitutes a new nerve fibre. Such an amputation neuroma is in reality a neurofibroma, for there is proliferation of connective tissue and new formation of nerve fibres.

A complete report of these cases is reserved for a later date.

January 13, 1898.

Aneurysm of the Arch of the Aorta.

S. McC. HAMILL, M. D.

I am indebted for the specimen shown to-night to Dr. John A. Johnston, of the National Soldiers' Home, of Virginia.

The history of the case, which is a very unsatisfactory one, is as follows: The man was admitted to the hospital on October 1, 1897. He had been found wandering around the post by one of the guards. There was no history obtainable. On admission his temperature was 104°F., pulse 130, respirations 40; he had muttering delirium and on examination was found to have a bilateral pneumonia. He gradually grew worse and died on the third of October. The autopsy was made thirty hours after death. The left lung was completely hepatized as was also the lower lobe of the right; the upper lobe of the right was acutely congested and there were three hemorrhagic infarcts on its upper surface. The heart and its aneurysmal aorta I here exhibit.

The heart is not much hypertrophied, rather less so than one would expect in view of the probable rather long standing of this case. There is some slight thickening of the posterior leaflet of the aortic valve, and there are a few deposits in the mitral leaflets.

The thoracic aorta is somewhat dilated from its very beginning, its walls are remarkably thickened and atheromatous. Beginning just between the left common carotid and the subclavian arteries the aorta suddenly expands into a huge cylindrical sack which extends almost throughout the entire length of the thoracic aorta. This sac is occupied by a very large organized clot, which is grooved anteriorly to admit of the passage of the blood. Over the site of the subclavian artery the clot is perforated and apparently allowed a free flow of blood to the arm. This is one of the most interesting features of the specimen. The sternum and those portions of the first rib and clavicle which came in contact with the sac were eroded.

DISCUSSION.

DR. RIESMAN.—The heart is obviously smaller than normal, which is contrary to general teaching at least, according to which atheroma causes cardiac hypertrophy, which may be further increased by the disturbances in the pressure of the main blood-stream due to the aneurysmal sac. I have, however observed, before, the absence of hypertrophy.

DR. COPLIN.—I have usually seen hypertrophy in such cases. It is likely due to artero-sclerosis; but in searching for that one must not be content with examining the peripheral vessels. The mesenteric vessels should be carefully inspected since, as Thoma has shown, atheroma is common in them.

DR. STEELE.—I recall a case of extreme atheroma of the arch and thoracic aorta in which the heart was not enlarged. Hasenfeld states that in cases of general arterial sclerosis in which the peripheral arterioles are not affected, there is usually little or no hypertrophy of the left ventricle. Such hypertrophy occurs only when the splanchnic vessels or the thoracic aorta are thickened, a statement that the case mentioned would seem to contradict.

DR. HUGHES agreed that cardiac hypertrophy is not the rule in aortic aneurysm, and expressed the opinion that sclerosis of the trunks has little to do with hypertrophy.

DR. HAMILL.—I have not noted hypertrophy constantly; in many cases a cardiac displacement is responsible for the idea.

January 27, 1898.

Syphiloma of the Liver.

H. M. FISHER, M. D.

The subject died at the Episcopal Hospital, November, 1897, of croupous pneumonia. The liver shows intense hepatitis (hepatitis gummosa). There are masses of granular shriveled cells in the centre of the foci. Surrounding these central cells is a zone of larger embryonic cells, many of which contain several nuclei. Between these cells is a fibrillar network. External to this zone are numerous small round cells (leucocytes) or nuclei. In the vessels, with the outer wall of which the gummæ are practically continuous, the endothelial cells of the intima have undergone enormous proliferation, and the flattened cells are so arranged, layer upon layer, that they almost block the lumen of the vessels. The centre of the nodules is almost amorphous and contains a few fine granules. Around the caseous mass is a distinctly fibrous zone. The fibrous capsule gradually shades off and sends out long radiating processes into the surrounding cirrhotic tissue, between the bands of which may be seen the atrophied liver cells. The caseous mass is made up of shriveled granular debris, into which run bands of fibrous tissue.

January 13, 1898.

Colloid Cyst of the Ovary, Involving the Splenic Capsule.

E. L. DUER, M. D., and A. E. BLACKBURN, M. D.

The patient, a white woman, age 57, was admitted to Presbyterian Hospital in December, 1897, under the care of Dr. Duer. Her sparse features were pinched, her expression was anxious, the skin was sallow, the abdomen was equally distended, and its superficial veins very prominent, the umbilicus pouting. The abdomen was not painful to the touch; fluctuation and dullness on percus sion were not present.

One and a half years ago she had noticed that her abdomen was enlarging. This progressed very slowly until September last, when it began to increase in size very rapidly. During the past nine months she has lost sixty pounds. At the operation, on opening the abdomen, a sac was found to be intimately adherent to the parietal peritoneum; incision of this revealed the peritoneal cavity filled with a papillomatous colloid mass, springing from the right ovary.

The growth apparently involved the spleen, which was removed, together with one and one-half gallons of the colloid masses, all of which are here exhibited.

DISCUSSION.

DR. STEELE.—The microscopic examination of the mass connected with the ovary shows it to be a cyst-adenoma papilliferum, which has undergone colloid degeneration. The substance of the spleen is not involved, but there are several sacs of the colloid material adherent to its capsule, and in two places these sacs have extended in along its trabeculæ. It is, however, easy to trace the capsule of the sac everywhere, and in no place is the splenic tissue degenerated. The involvement of the organ is therefore only apparent.

January 13, 1898.

Multiple Sarcomata.

FROM THE PATHOLOGICAL LABORATORY OF THE GERMAN HOSPITAL.

ALOYSIUS O. J. KELLY, M. D.

As a preliminary report, there were exhibited the organs from a patient who had just died of multiple sarcomata. The patient was a girl, aged 9 years. Eight months prior to her death her left thigh had been amputated at the hip for sarcoma of the femur. For a time subsequent to the operation she was in apparent good health, but latterly became much debilitated as a result of very manifest metastatic formations. The specimens exhibited were the organs of the thorax and neck, which revealed growths of the anterior, middle, and posterior mediastinal spaces, the lungs and the pleuræ; and in addition, the brain and the calvarium, showing growths arising from the dura, compressing the brain, penetrating through the skull, and elevating the scalp to a considerable extent.

January 13, 1898.

Renal Cirrhosis.

JOSEPH SAILER, M. D.

The specimens were obtained from a patient who was sent to the mortuary with the diagnosis of chronic interstitial nephritis and uremia. The subject was a white man of sixty years. The following are the notes of the post-mortem examination:

There was anthracosis of both lungs, with extensive pleural adhesions. The heart weighed 400 Grms. The tricuspid and mitral orifices were normal; the wall of the right ventricle was five mm. thick, the outer half being fatty. The wall of the left ventricle was twenty mm. thick, the muscle, as here seen, is reddish-yellow and shows a few yellow streaks. Two aberrant chordæ tendinæ are attached to the ventricular septum. The pulmonary leaflets are extensively fenestrated; the aortic valves were not competent, according to the hydrostatic test, if only a low pressure was used, but were perfectly competent if the pressure of the water was increased. The edges of the aortic leaflets are thickened, and there is a slight adhesion between the right and the posterior leaflets, and a small fenestration in the latter. An interesting anomaly is the communication of the posterior coronary artery with the sinus of Valsalva by two openings. The coronary arteries seem normal and the entire aorta was elastic. The right kidney weighed eighty-five, the left eighty Grms. Both are firm, reddish, the capsules are adherent, the cortices are thin and contain cysts. The wall of the bladder was thickened and rugous, and the middle lobe of the prostate was hypertrophied. The case is interesting as an illustration of the hypertrophy of the left ventricle of the heart, apparently without dilitation, in consequence of cirrhosis of the kidneys, since neither the valvular lesions at the aortic orifice, nor the condition of the aorta and peripheral vessels, would seem to have been sufficient of themselves to have caused this condition.

December 23, 1897.

Ecchinococcus Cyst of the Liver.

J. DUTTON STEELE, M. D.

The following specimens were obtained from a surgical operation performed a few days ago in a western town. No data can be given except that the material is part of the wall of a cyst that was removed from the liver of an adult.

The first slide shows very well the two layers of the cyst wall, with vessels and muscle fibers. Hooklets are present in large numbers, with here and there a scolex evidently broken loose from the breeding capsule; many of these show the rostellum intact, the four suckers and the sack-like hinder part of the parasite.

The second slide contains several peculiar bodies that are present in greater or less numbers in all the pieces of the cyst. These are pear-shaped bodies somewhat larger than the scolex, with a laminated structure, and in the glycerine preparation are quite opaque. They resemble a mussel shell in shape and in the arrangement of the markings. Mineral acids do not dissolve them although they have the appearance of being calcified. Acetic acid causes them to become a little more transparent than before and discloses a few round and oblong granular bodies within them. These last have no resemblance to the scolices. A search through the principal authorities throws no light upon the nature of these bodies. It is possible that they are budding "breeding capsules" though the density of their wall suggests rather a retrograde change. *December 23, 1897.*

Proceedings
of the
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MARCH 15, 1898. NEW SERIES, VOL. 1, NO. 5.

TABLE OF CONTENTS.

Symposium on the Pathology of the Diseases of the Cardio-Vascular System:—**HUGHES**, The Pericardium,—**MUSSER** and **STEELE**, The Myocardium,—**WILSON**, The Endocardium,—**PACKARD**, The Blood-vessels,—**STENGEL**, The Blood in Diseases of the Cardio-Vascular System,—**TAYLOR**, The Urine in Diseases of the Cardio-Vascular System.——**PACKARD**, Multiple Outgrowths from the Serous Surface of the Ileum.—**WILLIAMS**, A Tubal Gestation-Sac with Unruptured Membranes.—**HUGHES**, Carcinoma of the Common Bile-Duct and Head of the Pancreas.—**COPLIN**, Description of New Apparatus.—**SMITH**, Acute Epiphysitis and Purulent Pericarditis Complicating Typhoid Fever.—**SAILER**, A Case of Congenital Enteroptosis.—**EDSALL**, Typhoid Fever, with Irregular Lesions.

Symposium on the Pathology of the Diseases of the Cardio-Vascular System.

The Pericardium.

W. E. HUGHES, M.D.

In opening the subject of Diseases of the Cardio-Vascular System with this consideration of the pericardium, it would be well to premise by calling attention to the fact that the clinical significance of all such diseases is gauged largely by the effect they may have directly or remotely upon the heart muscle. Pathologically, the processes are governed by the same laws that hold in other systems. Thus, the pericardium being a closed sac, lined with a serous membrane, shows the same diseased conditions as obtain in other serous structures. But these conditions entail peculiar consequences, partly because the pericardium is such a closed sac, but principally because it is in such close juxtaposition to the heart muscle. Of the diseases of the pericardium that which is commonest and concerns us most closely, both pathologically and clinically, is unquestionably inflammation or *pericarditis*.

This may be divided, considering the nature of the process together with the results, into *fibrinous*, *sero-fibrinous*, *hemorrhagic* and *purulent*, or, considering the etiological factors, into *rheumatic*, *nephritic* and *specific* or bacterial. Clinically the results or effects of pericarditis are twofold : it has an immediate effect in its acuteness, largely mechanical; and a remote one, even after all inflammatory process has subsided, resulting in alteration of the structure of the heart. While at present only a limited number of cases of pericarditis can be classed as bacterial in their origin, yet that number has lately been increasing and it is possible that ultimately germs may be recognized as the causation factor in all. For the present it is necessary to retain the classification just given. The nature of the process, or rather of the resulting exudate, is important, at least immediately, for on that depends, aside from other considerations, the amount of mechanical interference with the heart action, and it is on that, as I have said, that the clinical effects of the acute disease largely depend. Further, in addition to the mechanical effect of the exudate, there must be taken into account in estimating the importance of the process, the association with it of diseases of the heart. In the majority of cases the lesion is not limited to the pericardium, but involves as well the endocardium, with all its consequences. More than this, and here probably are contained the gravest consequences of pericarditis, either from the same cause that produced the pericarditis or spreading inward from the inflamed pericardium, the heart muscle becomes involved in the same inflammation.

Where the exudate is fibrinous, and this is probably the commonest variety of pericarditis, it has comparatively little immediate mechanical effect, and these lack much of the gravity of some of the other forms. Where the effusion is more largely serous the gravity is increased, depending to a large extent upon the quantity of the effusion. Hemorrhagic pericarditis accompanies exhausting diseases and blood dyscrasiae, most notably scurvy, purpura, the low fevers, and is serious on account of the associated general condition. The process is in nowise different from that leading to sero-fibrinous exudates ; there is merely added to the inflammation a tendency to free hemorrhage. It is a rare variety. Purulent pericarditis, always bacterial in origin, is much the gravest form.

The effusion is large in amount, and while forming slowly increases steadily, and, having no vent, the pus, even though it may have had very little deleterious effect mechanically, eventually produces the same systemic state as any other undrained abscess. It is probable that small quantities of pus may be absorbed, but this must be an event of great rarity. Unlike an empyema, the pus in the pericardium shows little tendency to perforate and evacuate itself, so that spontaneous cure by this method is scarcely to be hoped for.

In the first three forms of exudate the tendency is toward the absorption of serum if this be present. The whole process of effusion and absorption may be, and in fact ordinarily is, a very rapid one, a week or even less usually sufficing for the beginning and end of an effusion which stretches the pericardium to its limit. The fibrinous element may organize into permanent adhesions, constituting a future danger, or it, too, may be completely absorbed. A sero-fibrinous pericarditis may run its course, then, and leave the pericardium with no recognizable lesion. Whether there be adhesions left or not depends in some inexplicable way on the nature of the process. The violence of the attack apparently has nothing to do with it. In the young these adhesions appear to be more likely to form than in later life. Of course the probabilities always are that a case of pericarditis will result in adhesions of greater or less extent.

In addition to the effusion there is probably always associated with the inflammation of the pericardium an acute myocarditis. In its milder forms this myocarditis is a direct consequence of the pericarditis and is limited in extent and will not involve the whole thickness of the heart muscle. In its graver forms it affects the muscle throughout, and is due to the same cause which produced the pericarditis. It is to this myocarditis that much of the gravity of pericarditis is due. As a result of it there is during the acuteness of the pericarditis a dilatation of the heart, which may end in any of three ways: death, complete restoration to a normal size, or permanent dilatation with hypertrophy. The amount of this myocarditis is governed by no apparent rule, nor can its extent be at all predicted from the nature of the case. Whether it entail serious future consequences or not apparently depends, at least in some measure, on the fate of the exudate. Where this organizes into firm adhesions the heart dilatation is likely to be permanent.

Rheumatic pericarditis is much the commonest variety. It is associated with rheumatism ordinarily early in the course of the attack, but may supervene at any time. The severity of the rheumatism has little to do with the incidence of pericarditis, many of the cases originating during scarcely noticeable rheumatic attacks. Classed with this variety may be the pericarditis originating during chorea. While pericarditis is commonest during the first rheumatic attack it may come on after any number. It affects males rather more frequently than females and most those engaged in laborious and exposed occupations, looking a little as though an interference with the elimination of the rheumatic poison might have thrown it back upon the pericardium. This is essentially an acute variety with rather prominent symptoms and is easily recognized clinically. The effusion, more largely fibrinous than serous, as a rule reaches no abnormally great degree and has a strong tendency to absorb perfectly. This form is, more than any other, attended with an acute myocarditis extending throughout the muscle, due to the rheumatic poison itself and not caused by extension inward of the pericardial inflammation. The immediate prognosis is quite good, very few of the cases dying in the acute attack, for, while the myocarditis is prominent, yet the effusion being limited in amount there is comparatively little crippling of the heart from external pressure. The ultimate prognosis is rather more doubtful, but still on the whole good, for it is only exceptional that firm adhesions form. It must be remembered that almost all cases have an associated endocarditis and it is rather on the degree and consequences of this that the prognosis depends.

In the course of Bright's disease pericarditis occurs with such frequency and this, too, without the intervention of any other exciting cause, that there can be no doubt of the relationship of cause and effect. Looking over the varieties of nephritis in which it occurs certain well-marked facts are strikingly suggestive. Thus, it is most common in the acute nephritis of adults, not of children, and in chronic interstitial nephritis. Then, too, it occurs with notable frequency accompanying uræmia. For the significance of these facts one must look to the extent of the toxæmia at the time of the occurrence of the pericarditis. Acute nephritis of adults is really not so much an actual acute disease as it is an exacerbation of a chronic condition and this exacerbation is coincident with, or dependent upon, an increase in the peculiar poisons in the blood incident to this disease. In the acute nephritis of children there is, if not less poison present, certainly greater rapidity of its elimination. Chronic interstitial nephritis is that form where there is a slow accumulation of poison with steadily failing eliminatory power. When pericarditis accompanies uræmia it might be said that it was primary, merely the disturber of balance and the producer of uræmia. But frequently the uræmia unquestionably precedes and is the direct excitant of the pericardial inflammation. Thus it may be argued that pericarditis occurring in the course of Bright's disease is directly owing to the local action of the poison circulating in the blood upon the pericardium. There will readily be traced here a striking analogy between this and rheumatic pericarditis, where the rheumatism furnishes the exciting poison and where, too, apparently a checking of elimination throws the poison more strongly upon the pericardium. Nephritis has another analogy here with rheumatism, in that not alone the pericardium but also other serous membranes are affected, but, unlike rheumatism, the pleuræ are more frequently affected than the pericardium.

Where micro-organisms are the cause of pericarditis they may be divided into common pyogenic organisms together with probably certain indefinite ones, pneumococcus and tubercle bacillus. This pericarditis may accompany any traumatism or constitutional pus infection, gonorrhœa or many acute diseases, most notably erysipelas and the eruptive fevers. It is graver prognostically than either of the two preceding varieties though fortunately less common. The effusion may be large and absorbed with difficulty or not rarely purulent and the tendency is to firm adhesions, ultimately crippling the heart. Of the bacterial varieties, the metapneumonic and tubercular are much the most important.

Pericarditis frequently complicates pneumonia, especially pneumonia of the left lung when the pneumococcus can directly from the diseased lung gain access to the pericardium. Even when there is no demonstrable pneumonia, but only some trifling and indefinite lung affection, the pneumococcus still may be the causative agent, and may be, equally as in frank pneumonia, provocative of pericarditis. Metapneumonic pericarditis has a peculiar significance in its ultimate results upon the heart. At the time of its acuteness it adds, as a rule, comparatively little to the gravity of the pneumonia, for, while there is usually an extensive myocarditis, yet the effusion is not sufficient in bulk to be harmful. Exceptions to this are those cases in which there is a large sero-fibrinous exudate, or when the exudate is pus, an event which occasionally happens in this form of pericarditis. It is the remote effects which are important. The fibrinous exudate has a more marked tendency than in most other varieties of pericarditis to organization and the formation of firm adhesions. Add to this the fact that not alone the pericardium, but the pleural and the mediastinal tissues, as well, are involved in the metapneumonic inflammation, and it can be easily seen how grave may be the outcome.

Tubercular pericarditis is recognized at the present day as a comparatively common form of the disease. It may be primary, in this case starting probably in the mediastinal glands, or secondary to pulmonary tuberculosis, or a part of general tuberculosis of the serous membranes. Usually chronic and with symptoms little marked, it may be acute and simulate any other variety. The exudate is principally fibrinous. Exceptionally, the exudate may be hemorrhagic, or even purulent. While the termination of the case is usually that of any tuberculosis, yet I have seen cases that have led me to suspect that tuberculous pericarditis, like tuberculous pleurisy, may recover perfectly and permanently.

Adherent Pericardium.—While the fibrinous exudate tends to be absorbed or to produce a few loose adhesions not at all hampering the heart's action, exceptionally these adhesions will be so firm and universal as to constitute an adherent pericardium, that is, a pericardial sac practically obliterated. Where this is the case, the heart is found to be hypertrophied and dilated, or normal, or atrophied. From the condition of the heart, it must be that the adhesions themselves have no necessary effect. Whether the heart be permanently affected or not, probably depends primarily on the state of its muscle. In many cases of acute pericarditis, most notably the rheumatic and metapneumonic, there is an accompanying acute myocarditis. This will necessarily entail a temporary dilatation, which may become permanent if the adhesions form rapidly enough. More important even than this acute condition is the fact that the fibroid pericardium is followed by a slowly ingrowing fibroid myocarditis, and it is this, rather than the acute myocarditis with temporary dilatation, that would determine the ultimate effect of adherent pericardium. This would explain the irregularity of the effect on the heart and why sometimes we have hypertrophy and sometimes atrophy. When the mediastinal tissues are involved, an indurative mediastino-pericarditis, the effect would be still more marked and constant, which is a clinical fact, for, from the nature of the process, there would be greater tendency to fibroid myocarditis as well as greater mechanical crippling power. In some of these cases more is involved than can be the mere result of an acute pericarditis ending in adhesions and involving the integrity of the heart; there is a widespread general process of which pericarditis is but a part or else the starting point. This is shown in those cases of adherent pericardium in which ascites is a prominent clinical feature. For the most tenable explanation of this ascites is that it is due to a slow change in the peritoneum similar in kind to the change in the pericardium, and not to any mechanical interference. I should suggest, then, two divisions of adherent pericardium: first, when there is no change in heart or when the change is brought about by valvular disease, and here the adherent pericardium would be a local process; second, when there is change in the heart secondary to the adherent pericardium; here the process is a general one, the mediastino-pericarditis being only a local manifestation. The cases in this latter division are oftener than is thought tubercular, and where not tubercular, will probably be found to be largely metapneumonic.

With the exception of this adherent pericardium, there are no serious effects left by pericarditis. The milk spots so frequently found on the surface of the heart are probably always the result of a pericarditis, or at least of a degenerative change of practically the same nature, but they are not important clinically.

The remaining pathological conditions of the pericardium can be dismissed in a few words.

Hydropericardium is usually a part of a general dropsy. Occasionally it is due to a local interference with the circulation. Very rarely, I think, it may be due to a slow change in the pericardium, which can scarcely be classed as an inflammation. Probably syphilis is at the bottom of these cases, if they exist, but of them I am not certain.

Hæmopericardium is due to the rupture of an aneurism of the aorta, heart or coronary artery, or to a rupture of the heart itself, rupture of an aortic aneurism being the most frequent cause.

Morbid growths are commonly secondary.

The Myocardium.

J. H. MUSSER, M. D. and J. D. STEELE, M. D.

Dr. Musser spoke as follows:—We wish, in opening, to recall your attention to the fact that it has taken physicians a long time to appreciate that valvulitis does not mean heart disease, and that it has taken longer to fully appreciate that even valvulitis plus dilatation does not mean heart disease. To explain, the congeries of symptoms to which the term heart disease should be applied—better still *morbus cardii,*—the valves, the heart muscle and the vessels must be included in the conception. The heart is a pumping station only in the circulation—itself presided over by a sensitive nervous mechanism,—at the other end of which is the great filter—the kidneys. We here exhibit specimens of various lesions of the myocardium, and beg to present our subject in this way.

Among other specimens is one of hypertrophy of the heart, in which vascular conditions were found which illustrate the relationship of the heart to the other parts of the circulatory apparatus.

The case was that of E. W., who had nephritis and cardiac hypertrophy, and died of apoplexy. At the autopsy the following was found:

The heart is much enlarged, its weight was 736 grms. The left ventricle was 28, the right ventricle 10 mm. in thickness. There are but a few spots of atheroma upon the aortic valves, but the aorta above the valves is excessively atheromatous with many calcareous plates. The coronary arteries are unusually healthy, with only a few scattered spots of sclerosis. The myocardium is much hypertrophied, but exhibits no scars or evidences of fatty degeneration. There are a few spots of atheroma at the base of the anterior leaflet of the mitral valve, which appears competent. The pulmonary and tricuspid valves are healthy and not dilated. The heart was empty. There was much sclerosis of the general arterial system. The central and basilar arteries are very much sclerosed; they stand out like tubes passing up into the Sylvian fissure. Numerous spots of atheroma are seen. There are several miliary aneurisms upon the branches of the Sylvian artery. A large hemorrhage is found in the right lateral ventricle, which has passed through the third and into the left ventricle, in which the blood is in a slightly fluid state.

The left kidney was 9 by 2 cm. and weighed 130 grms; the right was 10 by 4 cm. and weighed 140 grms. The capsules strip readily. The surfaces are pale and yellow, granular, and are scarred with a few cysts. In places, the organs are soft; in others hard. On section the cortices are soft and yellow, as a rule, though in places are many areas of congestion: they are distended—probably because of irregular areas of congestion. There is no congestion of the pyramids.

Sections from the apex of the left ventricle show considerable segmentation of the muscle fibre through the whole section. No other degeneration of the heart muscle is to be seen. The cells stain well and uniformly. There is some thickening of the intima of the smaller arteries, but to no marked degree. There is engorgement of the venous radicals. Sections from the kidney, show a chronic interstitial nephritis in a very advanced stage, with advanced degeneration of the renal cells and thickening of the intima of the arteries and arterial radicals.

We next exhibit the fresh specimens from a recent case of pernicious anæmia. The typical changes seen in the heart in this affection are well portrayed. During life the phenomena of extreme dilatation were witnessed, and after death this condition, as well as the fatty changes, were found.

The forms of myocarditis that occur in the course of general diseases as fever, intoxications, and the anæmiae, are both parenchymatous and interstitial, and their development into fatty, if it may be so termed, and fibrous myocarditis has been clearly traced. Cloudy swelling, granular and fatty degeneration, segmentation, and dissociation of the cells are here seen. Aneurism, and likewise dilatation of the heart, may be the result of this condition.

It is to the myocarditis succeeding coronary artery disease that we wish to devote special attention. Here local fibroid change or general fibrosis may follow; changes resulting from thrombosis of the coronary artery or its branches—ischæmia, and the inflammatory and degenerative changes resulting therefrom, as regeneration and overgrowth of fibrous tissue, and its consequent changes are the important lesions.

We exhibit several macro- and microscopical specimens, which were obtained from the following cases:

The first heart is from a woman of sixty-seven years, with valvulitis, myocarditis, and dilatation. The macroscopic appearances are described in Case III, in Dr. Musser's paper on angina pectoris (Trans. Asso. Amer. Phys., 1897, p. 70).

The right auricle is enormously dilated, and projects well to the right of the sternum; the left auricle is also greatly dilated, but not to the extent that the right is. The heart is much enlarged, and the apex extends to the left of the mid axillary line. The left ventricle contains some soft clots; it measures 20 mm. in thickness and 10 cm. in length. The myocardium is pale and friable; the papillary muscles show an exquisite mottling of yellow areas, the intermediate spaces being of a dark red. They appear almost cystic, and are so degenerated that they cannot stand upright by themselves.

The muscle of the ventricular wall shows numerous scars throughout its substance.

The leaflets of the mitral valve are thickened by extensive fibrous change; the edges of the leaflets are somewhat adherent; the chordæ are thickened and shortened; the valves admit two fingers to the first joint; the aorta above the valves shows considerable atheromatous change; the valves themselves, though slightly thickened, appear to be competent. The right ventricle is dilated; its walls measure 6 mm in thickness. The tricuspid valves are normal, but dilated, and admit five fingers to the second joint.

The weight of the heart is 480 grammes. The coronary arteries are exceedingly atheromatous and rigid, particularly in the upper portion; they do not contain thrombi, but are filled with marantic clot; the deposit of fat around them is somewhat excessive. The arteries throughout the body show a considerable degree of atheroma.

The kidneys are small; the capsule strips badly; there are numerous cysts on the surface of the cortex, which is irregular and scarred; the cortex is contracted, and the consistency of the organs is increased.

Sections from the *apex* show a moderate amount of new connective tissue formation forming little scars scattered here and there through the muscle. There is some fatty degeneration in the muscle fibres about these scars. The intima of the arteries is thickened throughout. Sections of the *papillary muscle* shows the above changes, but in much greater degree. The muscle of the cross-section is about one-third replaced by fibrous tissue, and the muscle remaining is degenerated in a remarkable degree. The cells are either much granulated or contain great vacuoles. The arteries are much thickened.

The second specimen was described in the same address. It was one of myocarditis and hypertrophic dilatation. The pericardium is normal. The heart is much enlarged. The right auricle is much dilated and extends to the parasternal line in the right. The myocardium of both ventricles is hypertrophied and both are dilated, the right especially.

The left ventricle measures 20 mm. in width. The right 8 mm. Weight of heart 480 grm. There are many scars throughout the myocardium of the left ventricle not localized to any one locality. The mitral valve is contracted, evidently by old inflammatory processes, and barely admits the first joint of the first finger. The tricuspid orifice is dilated and admits three fingers easily, and four to the second joint. The valves are slightly thickened along their free edge, but there is no evidence of acute inflammatory change.

The conus arteriosus of the aorta is contracted, evidently from a congenital malformation, and at its narrowest point (just below the valves) measures 15 mm. in diameter. The aortic valves are exceedingly thick and studded with calcareous nodules, but appear to be competent. The aorta is very much thickened, and there are calcareous plates about each coronary artery. The coronaries are thickened and calcareous throughout almost their entire extent. Dissection shows no thrombosis or embolism.

There was no evidence of passive congestion of the internal organs, the result of cardiac dilatation.

Pieces taken from the apex show a tremendous increase in connective tissue, forming great masses of fibrous tissue that run in bands through the heart muscle. There are columns of muscle fibres, caught in these scars, which are considerably degenerated. With this exception, there is not marked degeneration of the muscle and no segmentation. There is much thickening of the arteries and arterioles throughout all the sections examined, the fibrosis affecting particularly the intima.

The next specimen is from a case which has not yet been reported. The subject, H. A., was a man of fifty-six years. Death occurred in an attack of angina pectoris.

At the autopsy the heart was relaxed and not much above the normal size. The vessels (veins) on the surface were gorged with blood. The heart had evidently stopped in diastole. The aortic valves were competent to the water test. There are a few scattered spots of fibrous change in them, but it is not very noticeable. The aorta, down to within 5 mm. of the orifice of the coronary arteries, is remarkably healthy and smooth. But in a circle about each coronary is a plate of sclerosis, with considerable calcareous infiltration. The mouths of the arteries are rigid and calcareous, but the openings do not seem to be reduced in size. The anterior coronary artery is excessively sclerosed half-way down to the left apex, and is so infiltrated with lime as to be so brittle that it can be broken across. It is patulous, though its lumen is evidently somewhat diminished. The sclerosis stops quite abruptly half-way to the apex. The posterior coronary artery is very much thickened and calcified for 1 cm. from its orifice, which is rigid and calcified, though of the usual size. One cm. from the orifice there is a cheesy mass that fills the artery and is of a grayish color. It can be easily picked out with the point of the knife. Beyond this for 5 mm. the artery is completely obliterated—changed into a fibrous cord. After this for 1 cm. the artery is somewhat dilated, but excessively sclerosed and calcified and receives a large branch anteriorly, which is also somewhat sclerosed. This cannot be traced for any distance, but appears to run toward the anterior branch. After this the artery is again obliterated for 4 mm. and then appears fairly healthy, and can be traced to the apex of the right ventricle.

The myocardium is slightly hypertrophied, and the left ventricle measures 12 mm. in thickness. It is pale. No scars are visible to the naked eye in the wall or septum. There is a faint yellow striation at the base of each papillary muscle. The chordæ tendinæ of the mitral valve are slightly thickened and whitish, but not appreciably shortened. The anterior leaflet is considerably sclerosed, especially at the base, but not enough to have offered obstruction or to have caused incompetency.

The endocardium of both ventricles and auricles appears normal. There is an aberrant chorda tendina running from the base of one of the papillary muscles across to the septum about its middle. The valves and endocardium of the right side seem perfectly normal and not dilated. Thickness of right ventricle was 5 mm. The weight of heart was 336 grms.

Sections taken from the apex show: (1) Segmentation of the muscle fibres; the fibres are broken up to a considerable degree in many scattered areas; in other areas no such phenomenon is seen. (2) Areas in which the cells are pale and do not take the stain distinctly. No granules can be seen in them, but the appearance of such fields suggest commencing fatty change. In all other places the cells appear to be free from degenerative change to a remarkable degree. (3) Thickening of the small arteries and arterioles, especially seen in the arterioles, some of which are so thickened as to be almost obliterated. Such thickening occurs principally in the intima and is seen uniformally in all the vessels in the sections examined. The veins are dilated and filled with blood. There is no marked proliferation of connective tissue visible except that in a few places there are collections of round cells about the arterioles.

The general arterial system, as far as examined, in the thorax, abdomen and brain, is quite free from disease. The capsules of the kidneys strip with moderate ease. The surfaces of both are traversed by deep fissures, into which the capsules extend. These are quite regular, dividing the organ up into lobes, are free from evidences of inflammation, and are apparently congenital. The surfaces of the organs are slightly granular and of a deep red color. Their size is slightly above the normal. On section the cortices are considerably diminished in breadth, the ratio being about 1 : 4½. Both organs are intensely congested, the cortices being of a deep crimson color. The pyramids, which are plainly marked, are of a deep purple. The organs feel tense and swollen, and their consistency is increased. The right weighed 128, the left 148 grms. The ureters and pelvic viscera were normal.

In the kidneys the vessels, veins and arteries are engorged with blood in a remarkable manner. The arteries and arterioles are much thickened. There is a slight increase in the connective tissue about the vessels, and also about the glomeruli throughout the section. The cells of the tubules are considerably degenerated in certain well-defined areas though the nuclei in most of these stain well and the cell retains its shape. These are probably recent.

The dura mater is quite generally adherent to the skull, over the whole calvarium, by fibrous adhesions. The sinuses are normal. The skull cap is quite thick. The central and basilar arteries appear quite healthy, and no areas of sclerosis are seen. The dura mater is not thickened and, as far as can be seen, there are no evidences of inflammation or other disease in the membranes. The pituitary body appears normal.

The Endocardium.

J. C. WILSON, M. D.

From the etiological standpoint, the part played by bacteria in endocarditis is of overwhelming importance. It may be well questioned whether acute endocarditis, in practically all cases, is not due to the action of these agents; and as a large proportion of the chronic cases constitute a later stage of the acute process, they are to be attributed to the same factors. Endocarditis, thus considered, is not a primary, but a secondary, infection, and runs a course largely determined, as regards intensity, by the activity of the local process and the energy of the toxines elaborated in that process. We cannot, in this connection, consider especially the part played by the primary disease on the one hand, nor on the other hand associated conditions in other viscera, such as infarction, embolism, and embolic thrombosis, which are due to the endocarditis; nor is it my part, on this occasion, to dwell on the later changes in the valves. The mere allusion to these relationships serves to explain the frequency of endocarditis, to emphasize its importance, and to remind us of that inter-dependence of morbid processes which our nosological arrangements too often ignore.

The reaction of the endocardium to the irritation of bacteria deposited upon it by the blood occurs, in the vast majority of cases, in adult life upon the left side of the heart; in foetal life upon the right side. The mitral valves are more frequently involved than the aortic. Both valve-systems are usually first affected on the aspect facing the approach of the blood-current, namely, on the auricular surfaces in the case of the mitral, and on the ventricular surfaces in the case of the aortic valves, and in both instances not at the very margin, but back at the line of maximum contact. The resulting lesion consists in inflammatory infiltration of the endothelium, the formation of granulation tissue, coagulation necrosis, and the deposition of fibrin, often in layers. In the meshes of the masses thus formed are colonies of micro-organisms. These masses or vegetations in the less intense forms are minute and bead-like, presenting a warty or verrucose appearance. They are sometimes attached by narrow pedicles. In such cases they are usually limited to the reduplications of the endocardium which constitute the valves. In some instances the chordae tendineae are also involved. Such, briefly, are the lesions of *simple* or *verrucose endocarditis*. In the more intense cases extreme proliferation of the sub-endothelial connective tissue elements takes place, large masses of granulation tissue are formed upon which fibrin is deposited and in which extensive necrosis occurs, which extending leads variously to ulceration, the formation of pouches or valvular aneurisms, or to perforation or destruction of the valves. Here the process is not restricted to the valves, but frequently invades the adjacent endocardial lining of the chamber and causes ulceration, cardiac aneurism, or in some instances perforation of the wall of the heart; abscess-formation may also occur in the valve or heart wall. This constitutes *malignant* or *ulcerative endocarditis*. Between these two extremes there is no essential etiological or anatomical difference. It is simply a question of the virulence of the process and the extent of the lesions. The same micro-organisms are found in the benign and malignant forms, and the varying lesions are the progressive manifestations of degrees of intensity in the process from the lightest to the most severe. Simple endocarditis may at any time, by reinforcement or secondary infection, be rapidly converted into the malignant form. The vast majority of cases of endocarditis must be classed as simple or benign; the malignant cases are relatively few.

The usual termination of the benign cases is in resolution, which takes place by the cicatrization of the granulation tissue with the result, in many instances, of more or less extensive nodular thickening and deformity of the valve, the function of which is correspondingly and progressively impaired. The progress of the contraction under favorable circumstances is fortunately often slow. In other instances where the lesions have been more extensive the warty vegetations persist, undergoing sclerotic changes, the borders of the valve become adherent, lime salts may be deposited, and great deformity and loss of function result. The thickening, alteration in contour, and rigidity of the valvular mechanism result in valvular incompetence or narrowing of the orifice, or in a combination of these derangements. The condition resulting from cicatrization is described under such terms as *chronic*, *sclerotic*, *fibrous*, or *fibro-indurative endocarditis*—unfortunate terms, since the process is not an inflammatory, but a degenerative, one. Cardiac valves thus affected appear to be especially liable to subsequent attacks of acute inflammation, the evidences of which are often clinically present and demonstrable after death as *recurrent endocarditis*. In rare instances it is very probable, but not demonstrable, that the lesions of an acute endocarditis of minimum extent and intensity may undergo complete resolution, and that *restitutio ad integrum* may occur. This assumption has only clinical evidence in its support, and rests upon the acoustic characters of certain transitory murmurs which have been thought not to be ascribable to relative insufficiency or changes in the blood alone.

It is necessary to bear in mind that the growth and development of the colonies of micro-organisms which produce the endocarditis are accompanied by the formation of toxines, to the action of which the constitutional symptoms are chiefly to be ascribed. A relatively feeble intensity and perhaps smaller volume of the toxines produced in simple or benign endocarditis must be assumed in view of the insignificant character of the constitutional phenomena. In fact, in a large proportion of these cases general symptoms are almost wholly absent, and the antecedent disease runs its course unmodified, the intercurrent process being recognized by its physical signs alone. As in benign cases the acute lesions undergo resolution and sclerotic changes are established, the local and constitutional evidences of mechanical derangements of the circulatory apparatus appear; but these may be insignificant and easily overlooked for a long period of time. In the graver forms of endocarditis toxines of great virulence, and in larger quantity, produce a correspondingly violent constitutional reaction, and we see the clinical manifestations of a profound toxæmia, irregular chills, fever of high grade, with erratic fluctuations, profuse sweating, extreme asthenia, delirium, drowsiness, coma, etc.

Romberg[1] has suggested that certain of the symptoms of acute endocarditis are due to myocardial defects, and has reported two cases of verrucose endocarditis of rheumatic origin, in which acute interstitial myocarditis and hyaline thrombosis of the small blood-vessels of the myocardium were found. These changes were most marked near the auricular border.

The organisms which have been obtained in culture from the vegetations in endocarditis are numerous. Osler[2] enumerates streptococci, staphylococci, the more common; the micrococcus lanceolatus, the bacillus typhi abdominalis, the bacillus tuberculosis, the gonococcus, and the bacillus anthrasis. Oertel[3] gives the following list: Staphylococcus pyogenes aureus et albus, staphylococcus cereus albus, staphylococcus flavus non pyogenes, bacillus pyogenes fœtidus, streptococcus pyogenes, diplococcus pneumoniæ, gonococcus of Neisser. In the course of the last half decade the gonococcus of Neisser has been found in many instances in the lesions of malignant endocarditis in individuals giving a history of recent gonorrhœa. The early researches of Councilman are of importance in this connection. Howard[4] has reported a case of endocarditis due to the bacillus diphtheriæ. Wright and Stokes[5] isolated in seven out of ten cases the micrococcus lanceolatus from the vegetations or ulceration of the heart, and in one instance this micro-organism was associated with the bacillus diphtheriæ. Charrin[6] has reported an interesting case of endocarditis of the right side of the heart in a male of eighteen years. Exuberant vegetations were present upon the pulmonary valve and upon the pulmonary leaflets. In these vegetations the staphylococcus aureus was found. The same organisms had been very abundant in the secretion from the tonsils. The patient had suffered from an attack of influenza, followed by extensive broncho-pneumonia. Numerous investigators have discovered the tubercle bacillus in the lesions of ulcerative endocarditis. Leyden[7] recently found this organism in the vegetations in four cases of acute endocarditis, in one of which the bacilli were also present in antemortem clots in the heart. This observer ascribes the endocarditis not directly to the tubercle bacilli, but to other pathogenic organisms present.

The diseases with which endocarditis is especially associated are: (*a*) With great frequency, but commonly in the benign form; rheumatic fever, peliosis rheumatica, chorea, and angina tonsillaris. (*b*) More rarely, and usually in the benign form: scarlet fever, measles, influenza, and enteric fever. (*c*) Also rarely, but then often in the malignant form: pneumonia, pleurisy, diphtheria, the variolous diseases, acute and chronic nephritis, carcinoma, and various tuberculous processes. (*d*) Finally, and almost always in the malignant form: all grave septic and pyemic affections, especially those that occur in the puerperium, pylephlebitis, caries, and in particular that of the temporal bone in connection with ear disease, infected wounds, often apparently trifling in themselves, and the like.

(1) Deutsch. Archiv f. Klin. Med., 1894. (2) Practice of Medicine, second edition, 1895. (3) Encyklopædie der Therapie, Band ii, 1897. (4) Am. Jour. Med. Sci., Dec., 1894. (5) Boston Med. and Surg. Jour., 1895. (6) Semaine Med., Mar. 14, 1896. (7) Deutsch. Med. Woch., 9 Jan., 1896.

The Blood-vessels.

FREDERICK A. PACKARD, M. D.

The subject of the pathology of the arterial system is so broad that I have decided to give only a brief summary of the more important contributions to our knowledge of diseases of the blood-vessels that have been offered during the past year or two, since I feel that some of them have been of great value in advancing our knowledge.

When we consider that we grow old at a rate commensurate with the pathological changes in our arteries, and that our knowledge of senescence is still far from complete, the importance of more complete studies of the pathology of the arteries is readily manifest. Two thoughts I would suggest at the outset. One of these is that there is a close analogy between the functions of the heart and of the arteries on both physiological and embryological grounds. Taking the simplest form of heart, we find it merely a specialized portion of the vascular tube, just as it forms really a specialized spot in the vascular system of the growing embryo. We are rather too apt to think of the arteries as simply conducting tubes whose sole function is the passive one of forming a communication between the heart and the various tissues and organs. From a physiological, diagnostic and therapeutic standpoint this gives far too narrow a view of their duties. Another point to which I would allude is the disadvantage under which the arteries suffer in being open to insults from two sources, the blood within their lumen (which may work harm both physically, from its distending force, and bio-chemically, from the noxious materials which it may contain), and also the nutrient blood furnished to their walls by the vasa vasorum.

Another thought that is, I think, worth mentioning, was suggested to me by a case that occurred in my ward at the Philadelphia Hospital some years ago. The man had murmurs, evidently not of cardiac origin, over many of the chief arteries of his body, during the course of an acute febrile attack with wild delirium. These murmurs disappeared with convalescence, and I have always felt quite positive that he had acute generalized endarteritis. I can see no good reason to believe that the endarterium is not as subject to inflammation as is the endocardium.

During the past year three articles of distinct importance have appeared in regard to acute aortitis. In the *Bull. et Mém. de la Soc. Méd. des Hopitaux* for October 28, 1897, Thoinot and Griffon report the case of a woman aged fifty-three years, who had suffered from facial erysipelas in March, 1896, which attack was followed by insomnia, general weakness and dyspnœa. For these symptoms she was admitted to hospital on March 27. On the day after admission she had a relapse of erysipelas, with slight local but marked general symptoms. Four days later dyspnœa increased, and there was a murmur occupying the time of the second sound and transmitted down the sternum. After continuing for a few days the general symptoms subsided, and she left the hospital on April 22, only to be readmitted in eight days for increasing weakness and dyspnœa. Symptoms of infection became more marked, and she died almost a month after her final admission. At the autopsy ancient and recent lesions were found in the aorta, the latter characterized by fibrinous, round, gelatinous deposits of amber yellow color and slightly elevated above the surface. These were present only in the ascending aorta and in the arch. These areas were found to involve the internal tunic, and especially the deeper portions of it. The plaques were formed essentially of fibrillary substance running parallel to the axis of the vessel, and large cells having the same direction. In the deeper portion of the plaques there was fibrillar tissue without much young cellular infiltration. Masses of cells penetrated perpendicularly into the internal coat in columns as though arranged about a blood-vessel. In the middle tunic they found, at corresponding points, degenerative changes with nuclear fragmentation and vessels running toward the internal coat. In the adventitia at points adjacent to the plaques on the intima, there was intense congestion, with many dilated capillaries, about which were masses of round embryonal cells. These masses of round cells formed the columns projecting through the media into the internal tunic.

Iselin reported to the Societé Anatomique at its meeting on October 8, 1897 (abstract in *Gaz. hebdom. de Méd. et de Chir.*, October 14, 1897, p. 981), the case of a man aged fifty-four years, who was suddenly seized after effort by pain in the base of the neck and intense dyspnœa. No cardiac, renal or pulmonary cause for the dyspnœa could be found. His whole left arm was pulseless. He died suddenly, and at the autopsy the aorta was lacking in suppleness, the left sub-clavian artery was completely obstructed by a clot extending from its origin to the vertebral orifice. The whole thoracic aorta was the seat of acute aortitis, with thickening to the extent of 3 mm. The other organs were normal, except for early lesions of tuberculosis at the apices.

An important experimental contribution to our knowledge of the pathology of the blood-vessels has been made by Boinet and

Romary (*Archives de Méd. experiment. et d'anatomie pathologique*, 1897, t. ix., p. 902). Before proceeding to the experimental results which they obtained they report three cases with acute aortitis. Their first case was one of facial erysipelas, with four small rose-colored gelatinous plaques on the internal surface of the ascending aorta. These were formed by infiltration of the middle and deeper parts of the endarterium by embryonal cells. Streptococci in large numbers were found deep in the endarterium, in small numbers in the other tunics, and about the vasa vasorum. Their second case was one of rheumatic pericarditis. On the internal surface of the ascending aorta there were three gelatinous plaques containing cocci similar to those described by Boinet in 1891 as occurring in a case of acute articular rheumatism. These were found to be most numerous in the deep layer of the endarterium, but were also found in small quantities in the other tunics. Their third case was one of influenzal broncho-pneumonia. In the small fibrinous vegetations upon atheromatous ulcers of the aorta, and in the embryonal tissue which had invaded the deep layer of the endarterium, small cocci were found. After giving an historical review of the work done by previous investigators, the authors give their own experiments. In their first series of experiments they sought to determine the rôle of micro-organisms injected into the blood in producing aortitis after aseptic trauma of its intima. Injection of Eberth's bacilli into the marginal vein of the ear produced arteritis, as evidenced by two plaques of endarteritis and abundant infiltration by embryonal elements about the vasa vasorum of the external tunic in the descending aorta—a point not susceptible of being reached by their previous injuring of the aorta by way of the carotid. Bacterium coli commune produced aortitis at the area injured. The same was true of streptococci. On the other hand, the injection of staphylococcus after previous aortic trauma was not followed by aortitis. Injection of anthrax bacilli, which was followed by death in forty-eight hours, produced early changes of aortitis with bacilli present in the inflamed areas in the deep layers of the internal tunic. By injection of tubercle bacilli, whose activity was hastened by later injections of tuberculin, aortic lesions were produced at the seat of trauma. In order to determine whether bacterial products could alone produce aortitis they employed organisms whose wanderings are more or less restricted to the point of entry. A guinea-pig was injected with a pure culture of the tetanus bacillus. This was repeated in four days, and in order to increase their virulence streptococci were injected simultaneously. In the arch of the aorta lesions of aortitis were found which were free from tetanus bacilli. After subcutaneous injection of diphtheria bacilli, after previous trauma of the aorta, lesions of aortitis were found not only where the trauma was induced, but also in the descending aorta, which must have been uninjured by the scraping.

In order to determine the influence of bacterial toxines in the production of aortitis, they injected toxines of Klebs-Loeffler bacillus and streptococcus, and tuberculin. They found that the toxine of diphtheria of itself sufficed to produce aortitis without previous trauma. The same was true of the toxine of streptococcus. Tuberculin produced aortitis at the points previously injured. The toxine of cholera produced plaques of aortitis at the point injured by the stylet.

In order to determine the rôle of intoxications in producing aortitis they administered to animals white lead, producing aortitis, which was apparently aided by previous trauma; and uric acid and urates, which produced slight changes without, more marked changes with, prior trauma. The repeated subcutaneous injection of phloridzin (causing diabetes mellitus) produced aortitis, which was more intense when tuberculin was also injected. Repeated subcutaneous injections of glycerophosphate of mercury caused aortitis, which was most marked in the external tunic.

With both infections and intoxications capable of doing so much, is it any wonder that people grow old!

In the *Archives de Méd. expérimentale et d'anatomie pathologique* for 1896 (vol. viii, p. 784), Leopold Lévy reports a case in which he found in the subendothelial layer of the aorta a solitary rounded, grayish tubercle of the size of a pin-head, containing in its interior no blood-vessels, but a few giant-cells and tubercle bacilli. No other aortic lesion was found except for a small inflammatory nodule in the external tunic.

Strœbe, of Hanover (*Bericht über die Verhandlung der path.-anat. Section der 69. Versammlung deutscher Naturforscher und*

Aerzte zu Braunschurig vom 20. 25. Sept., 1897; Abstract in Centralbl. f. allg. Path. u. path. Anat., November 1, 1897, p. 866), has reported a case of generalized miliary tuberculosis occurring in a boy, aged sixteen years, in whose ascending aorta there was a polypoid growth 7 mm. long by 3 to 4 mm. thick, springing from the intima. The polyp was caseating at its centre. Numerous tubercle bacilli were found in the growth. Infection was supposed to have come from cheesy bronchial glands.

Two valuable articles upon atheroma and its consequences have been published in the last two volumes of the *Journal of Pathology and Bacteriology.* One of these is by W. Ainslie Hollis, the other by Joseph Coats and A. G. Auld. In the former paper there is mentioned the case of a child, dead four days after a severe burn, in whose aorta there were found streaks of early and evidently recent atheroma. Beside many other interesting points regarding the nature of atheroma and its consequences, the authors of the latter paper give an interesting account of the structure of minute pouchings of the aorta and early aneurisms. They especially insist upon atrophy of the media, which is accounted for by attrition upon that tunic by the atheromatous nodule in the intima, cerebral aneurisms being considered by them as being usually due to incomplete embolism, the emboli sticking to the vessel wall, and by constant slight attrition injuring and so weakening both intima and media.

In the Glasgow *Medical Journal* of August, 1897, Professor W. T. Gairdner has put upon record, and in the discussion upon his paper others mentioned, many examples of aortic aneurisms bulging into the pulmonary artery. In some of these cases there was a communication between the two vessels.

Lamplaugh (*Brit. Med. Jour.*, Aug. 14, 1897, p. 392), has recorded a case of a similar kind, and gives an analysis of fifteen cases from the literature.

In an article by A. Fraenkel (*Deutsche Med. Woch.*, February 4 and 11, 1897, pp. 85, 103), two cases of perforation of the descending vena cava (one of which was confirmed by autopsy) are added to the list of examples of that accident. In the same article he emphasizes the occurrence of indurative pneumonia as a result of pressure by aortic aneurism upon the left bronchus.

In an analysis of thirty-two cases of ruptured thoracic aneurism among 4593 autopsies, T. M. Kelynack (*Lancet*, July 24, 1897, p. 193), gives the statistics as to the point of rupture. The pericardium heads the list with 13 to its credit; the left pleura was the seat in 7; the œsophagus in 3; externally in 3; the trachea in 2; the right pleura, the left lung, the right bronchus and the superior vena cava, of each 1. G. Bertram Hunt (*Trans. Clinical Society of London*, 1897, vol. xxx, p. 200), reports a case of rupture into both pleural cavities, into the left (from which thirty-three ounces of blood were withdrawn by aspiration) thirteen days after the sudden onset of severe chest pain; into the right pleural cavity nine months later after an interval of comparative health. The last rupture abruptly terminated life. In this case the aneurism was of the dissecting type, and the author gives a good argument for the possible recognition of the condition by the physical signs and symptoms in his patient.

In the *Bull. et Mem. de la Soc. Med. des Hôp.*, of October 21, 1897, Thoinot and Bernard record a case presenting a rather rare point of rupture in the case of disease of the aorta. The hemorrhage took place from a rupture in the wall where there was a calcareous plate. The blood poured out between the tunics of the aorta and into the neighboring tissues of the mediastinum and 1700 cu. cm. had burrowed under the left costal pleura, forming a huge subpleural hæmatoma. Five other cases from literature having a similar location of the main extravasation, are collected by the authors. In three of the total of six cases dissecting aneurisms were present. In two there was said to be no sign of atheroma of the aorta. In four of the six the left pleura was the one dissected by the blood.

Regarding rupture of the aorta Delano Ames and W. Guy Townsend have written a valuable article in the *Maryland Medical Journal* for July 3 and 10, 1897. They analyze fifty cases, including dissecting aneurism, in addition to adding a case personally observed by them. In their case the aorta was very atheromatous. Of the fifty, in twenty-seven rupture took place through all coats at once, in six through intima and media primarily, and later through the external tunic; in eight there was dissecting aneurism. It is to be remarked that in a great number of the patients who survived the rupture for a sufficient length of time, the severest pain was referred to the abdomen. This also was the case in the patient whose aorta I present to-night. In eight per cent of the cases there was no atheroma.

In a monograph by Lesage (*De la mort subite dans l'aortite ou ses complications*, Paris, 1896), there is much of value regarding rupture of the aorta.

P. Bronardel (*La mort et la mort subite*, Paris, 1895), quotes a case previously reported by him where the non-atheromatous aorta of a student of medicine ruptured in two places, with left subpleural hæmatoma, as in the other cases recorded by Thoinot and Bernard.

Cases of obstruction of the abdominal aorta have been recorded by Charrier and Apert (*Bull. de la Soc. Anatomique de Paris*, 1896, 5*s*., t. x, p. 766), who also add ten other cases from literature; by R. P. Goodworth (*Brit. Med. Jour.*, June 20, 1896, p. 1501) and by Walter Bell (*Brit. Med. Jour.*, October 24, 1896, p. 1227). In the *Deutsche Medicinische Wochenschr.* for October 15, 1897, p. 672, Carl Helbing details the spinal cord changes from a case of thrombosis of the abdominal aorta In his case death occurred thirty-nine days after the appearance of gangrene, and he attributes the spinal cord changes to the natural amputation rather than to interference with the spinal blood supply.

Within the past two years several cases of communication between thoracic aneurism and the superior vena cava have been recorded.

I am glad to be enabled to show to the society specimens illustrating some of the rarer lesions spoken of above. These are simply shown as "card specimens," and will be the subject of a fuller report at a future time. Among these specimens are illustrations of recent atheroma, old atheroma, rupture of a non-atheromatous aortic arch, thrombosis of the abdominal aorta, and a specimen of aneurism of the aortic arch with gangrene of the left bronchus, and indurative fibroid pneumonia, such as Fraenkel has particularly emphasized.

The Blood in Diseases of the Cardio-Vascular System.

ALFRED STENGEL, M. D.

I cannot undertake a thorough discussion of this subject from every point of view, but wish to allude only to some of the more important questions suggested by the study of the blood in cardio-vascular troubles. I may divide the subject into the study of the blood (1) in acute inflammatory lesions, such as endocarditis and acute arteritis; (2) in atheroma and arterial degenerations; (3) in the case of mechanical lesions, such as those of chronic valvular disease.

(1) *Endocarditis.*—In acute endocarditis, either of the benign or malignant type, it is customary to find a rapid reduction in the number of red corpuscles. In severe cases I have found counts of 3,000,000 and even 2,500,000. There is in this no condition peculiar to the lesion, but merely the evidence of general infection. When the acute lesion produces valvular deficiencies or obstruction, the condition of the blood may be complicated by the existence of such valvular disturbances. The leukocytes in acute endocarditis are habitually increased in number, this also being the result of the infectious character of the disease. In one of my cases the differential count of the leukocytes was of interest from the inordinate proportionate of polymorphous cells. In a count of 500 leukocytes 98.5 per cent were of this description. Bacteriologic examination of the blood is of great importance in acute endocarditis, and may be the means of arriving at a diagnosis in doubtful cases. Various organisms have thus been obtained directly from the circulating blood and have afterward been found in the cardiac vegetations. Among others, the gonococcus is important in this connection. It has been obtained from the blood in pure cultures in several instances of supposed gonorrhœal endocarditis.

(2) *Atheromatous and Degenerative Conditions of the Vessels.*—The examination of the blood does not often present features of importance. It may be possible in certain instances to discover, from time to time, particles of detritis, fatty matter or other foreign bodies, which have been added to the blood from the lesions of the arteries. Such occurrences, however, are too rare to be of practical value. High grades of anæmia may be present in arterial disease,

but I have frequently been struck by the fact that the character of the blood was but moderately altered from the normal, though the appearance of the patients suggested marked anæmia. This may possibly be explained by the changed conditions in the peripheral arterioles.

(3) *The Blood in Valvular Disease.*—A number of investigations have been published regarding the blood in valvular disease of the heart, and most investigators have reported the discovery of a tendency to inspissation with increased number of red corpuscles. This has not, however, been the uniform result, and every one who has made a study of the subject has noted that changes are prone to occur from time to time in any case. This is particularly true of diseases of the mitral valve. It has been shown by Ottolenghi that reductions of blood pressure cause a diminution in the number of red corpuscles of the circulating blood and a dilution of the blood, in consequence of the entrance into the circulation of greater or less quantities of lymph; and it has been found by Grawitz and others that in chronic cardiac diseases attacks of failing compensation may be marked by such dilution of the blood and reduction in the number of corpuscles. This, however, is not constant. I have myself seen it in very marked degree, but, on the other hand, have seen cases in which decided loss of compensation led to a rapid increase in the number of corpuscles, in one instance amounting to 500,000 per cubic mm. Probably loss of compensation with reduction of blood pressure in itself might have this diluting effect, but the pathologic conditions in individual cases are so apt to be complicated by other elements of importance that uniform results are not to be expected.

In cases of chronic cardiac disease with continuous slight inadequacy of the circulation, an increased number of red corpuscles or polycythemia has been frequently detected and is of very great interest. It is doubtless in some way analagous to the increased number of corpuscles found in the blood of persons residing at high altitudes, and has been so considered by some. The degree of polycythemia may be very slight, but in some, notably in cases of congenital heart disease, the number of red corpuscles may run as high as 8,000,000 per cubic mm. A number of explanations have been offered for this condition. Among others, Grawitz is a recent defender of the theory that disturbance of the distribution of water in the system is an important causal element. He believes that in consequence of the continually reduced blood pressure and vascular dilatation there is loss of liquid from the blood, especially through the lungs, and that inspissation of the blood occurs in this way. I cannot now enter at length into a discussion of the experimental side of this question, but would only note certain objections to this theory. It has been shown that loss of water from the system by evaporation or through liquid discharges, affects the liquids of the body generally rather than the blood alone, and that with the knowledge we have regarding the interchange of liquids between the circulating blood and the tissues, we may reasonably conclude that notable changes in the blood would not ensue from such causes as Grawitz suggests. Further, the rapid fluctuations and the failure of polycythemia to occur in some cases in which all the conditions are present for such evaporation or loss of water as Grawitz maintains, show that the importance of this matter has, to say the least, been overstated. Finally, if the analogy with the conditions of the blood in persons at high altitudes, or in experiments on animals subjected to rarefied air be allowed to be sufficiently close to warrant deductions, it might be pointed out that the rapid fluctuations in the number of corpuscles would militate against the belief that loss of water from the system is the important cause.

Another theory, particularly to explain the increased number of corpuscles at high altitudes, is suggested by Koeppe. According to him there is probably a rapid formation of corpuscles by division of existing red corpuscles, as he was able to demonstrate experimentally after venesection. It is hardly likely, however, that this theory expresses the real causation of the change in the number of the corpuscles. The fluctuations are too rapid in both directions to warrant this view.

For my own part it has seemed to me that disturbances of the distribution of the corpuscles play a more important rôle in effecting alterations in the apparent constitution of the blood than investigators have generally been disposed to concede. It has long been known that the number of red corpuscles found in a cubic mm. of blood drawn from the skin is greater in dependent parts of the body than in elevated portions. Some years since I made some investigations of this matter, counting simultaneously the blood of the toe and of the finger held in air. These studies showed that the number of corpuscles in the blood of the toe was uniformly greater than that in the finger, the average excess being as much as eight to ten per cent. The explanation which seems reasonable in such instances is that the serum is more readily propelled against the force of gravity than the heavier corpuscles and that in consequence a greater number of the latter are retained in the circulation of dependent parts. Some support is furnished to this belief by the studies of J. K. Mitchell upon the influence of massage upon the constitution of the blood. His studies show that the number of the blood corpuscles progressively increases after general massage and I agree with him that this is probably due to the introduction into the circulation of considerable numbers of corpuscles which had previously rested in the tissues. Now in chronic cardiac diseases there are elements which would tend to cause a lagging of red cells in the peripheral arterioles or venules. Among these the failing cardiac power is most important. It is not improbable, however, that evaporation such as Grawitz urges may contribute to the result by a general inspissation of the blood, though it does not seem likely that this element is of more than secondary mportance. Another factor has suggested itself to me in the course of some studies of the changes occurring in red corpuscles in consequence of congestion. I found that the blood drawn from a finger firmly ligatured until it became deeply cyanosed underwent marked changes. I need not now repeat in detail the changes observed but desire to call attention to one fact alone, viz., that there was evident increase of the viscosity. This is but one stage or element in the process of progressive degeneration of corpuscles. It occurred to me that a similar change of less pronounced degree, however, might result from general congestion and inadequate oxygenation of the blood. Such a condition could conceivably exercise some influence in deterring the return circulation from the tissues, particularly when the vis a tergo is diminished. Another change observable in the corpuscles of congested parts is increase in size, at least it has seemed to me so in the few and rather imperfect tests I have made, by determining the number of corpuscles and the bulk as compared with the serum. A similar increase in size has been described as one of the conditions found in chronic cardiac disease. This also might be an added cause of peripheral corpuscular congestion. It seems to me then that the causes of polycythemia most likely operative in chronic cardiac disease are failing circulation, increased viscosity and possibly increased size of the red blood corpuscles.

This theory would, to a certain extent, be found compatible with the rapid fluctuations presented by some cases as well as the different behavior in aortic and mitral valvular disease. It has been my experience, as it has been that of others, that the blood in aortic valvular disease especially aortic regurgitation, is more commonly anæmic than in mitral disease. In aortic regurgitation the dilatation of the arterioles may possibly explain this. Given a certain degree of anæmia if the arterioles are widely distributed and the force of the heart rather great, peripheral stasis of the corpuscles would not be likely to occur; while in mitral cases with a similar degree of anæmia but without the arterial dilatation and with less adequate central power, the corpuscles might be arrested at the periphery and the blood might seemingly be of great richness though in reality no better constituted on the whole than in the other instances.

The studies of the solid matters of the blood and of the relation of serum to corpuscles so closely coincide with the results of the counting of the corpuscles, that I need not enter into them at the present time.

The leukocytes in chronic valvular disease are frequently increased in number, but the degree of leukocytosis is rarely great and the conditions are by no means uniform. The chemical studies of the blood and particularly the studies of the alkalinity and gaseous constituents furnish an inviting field for investigations, but thus far there have been few practical results.

The Urine in Diseases of the Cardio-Vascular System.

A. E. TAYLOR, M. D.

The alterations in the urine in the various acute diseases of this system are so extensively and obviously effected by other factors, as sepsis, toxines, fever, etc., that it would be almost hopeless and highly unprofitable to attempt to detail them here. I shall therefore confine my remarks to the chronic diseases of this system. The alterations may be briefly summarized as follows, all alterations due to renal diseases having been excluded.

Quantity: The volume of urine secreted in conditions of simple cardiac hypertrophy without vascular sclerosis, and in chronic valvular disease with compensation, is normal. Following broken compensation oliguria is the rule, but there may be periods of inexplicable polyuria. In arterosclerosis polyuria is the rule, especially with normal or increased ventricular power. Whether a general arterosclerosis which does not involve the renal vessels can produce polyuria is not clear; if the renal vessels were not altered in their lumen polyuria would probably ensue, but experimental work in other lines leads us to conclude, reasoning by analogy, that the kidneys possess vasomotor regulations which would permit them to so adjust themselves in relation to the increased blood-pressure as to maintain the normal output. The quantity also depends on other factors. If with cardiac disease there is marked respiratory distress, sweating is often excessive, induced apparently by the intimate reflex relations existing between the skin and the lungs; this reduces the secretion.

The total molecular output in the urine of most cases of valvular disease without compensation is reduced, often markedly. This is a most important fact. When we consider that these molecules, normally excreted but here returned to the venous blood, when estimated according to the laws of osmotic density, correspond to so and so many foot pounds of energy, it becomes apparent that such a state of affairs must profoundly modify the vascular and lymphatic circulations, and that the importance of this fact for the question of transudation must be recognized.

The nitrogen output is not known to be abnormal in these cases; acute dyspnœa in animals produces an increased output, due to an increase of proteid disintegration; this has never been shown for chronic cardiac dyspnœa. The nitrogen output obviously diminishes with the advent of or increase in dropsy; when the transudates are being eliminated it is correspondingly increased, and the high output is maintained several days after all traces of dropsy have disappeared. The urea is decreased in similar conditions more markedly than the other nitrogenous substances, so that the various urinary quotients are modified. It seems to be simply a question of nitrogen-retention, not of metabolism. The elimination of uric acid is within the normal limits; the precipitation of the urates so commonly seen in such urines is purely a physical phenomenon, and is not due to an excess of uric acid. The oxalates, ammonia, kreatinin and aceton are present in normal proportions. The phosphates are not in excess; the acid phosphates have been found relatively increased. That the urine in these cases is hyperacid is a statement founded, so far as I am able to determine, upon no exact estimations. I know of no accurate studies upon the salts; here again the question of dropsy would so complicate the circumstances as to render the results of doubtful value. Lactic acid is present in a large proportion of cases of chronic heart disease; it is not clear whether it owes its origin to hepatic congestion or to muscular disturbances.

Albuminuria in cardio-vascular disease can arise in several ways. Dyspnœa alone can produce it; later the system seems to become accustomed to dyspnœa and the albumen disappears from the urine. Dropsy per se seems able to produce it. Whether this can be held to be due to lymphatic transudation into the urinary tract or not I do not know, but one can often note a reduction in the albuminuria when dropsy is relieved by puncture or tapping, and may on the contrary note an increase when with compensation the dropsy is being freed by diuresis. Lastly congestion of the kidney produces albuminuria. It must not be assumed that any and all congestions cause it. At the onset of congestion albumen escapes in the urine, but soon the renal cells seem to accustom themselves in some way to the new conditions, and then perhaps for a long time no albumen passes into the urine. Many instances of congestion in cardiac disease are surely accompanied by cellular disease in the tubules. In cases of pure congestions the albuminuria is now explained as a transfusion, which mode of origin from the lymphatic system in dropsy as above alluded to, is therefore made probable. Serum-albumen and globulins are the only forms of proteid eliminated; the quantity is usually small, from ¼ to 2 G. per day. In amyloid disease the proportion of globulins is largely increased above the proportions seen in simple albuminuria.

Whether albuminuria appears in arterosclerosis without renal disease or congestion is doubtful.

Sugar and the Diazo-reaction are occasionally present, the explanations have not been furnished. Hydrobilirubin appears in the urine of many chronic cases, due to what is now called pleiochromia, the result of excessive blood-destruction, and to hepatic disturbances; this coexists with normally colored stools, and with the faint icteroid tint so often seen in the conjunctivæ and skin of such patients.

Many studies have been carried through with relation to the urinary toxicities in connection with heart diseases. The methods do not inspire confidence, and the results have not been such as to warrant their introduction into this discussion.

DISCUSSION.

DR. HERRICK, of Chicago, by invitation.—I beg to present the brief details of a case I have recently seen, in order to emphasize the fact that as a sequence of pericarditis adhesions may form between the outer pericardiac surface and the mediastinal structures. Following the typical course of pericarditis in this case, and in connection with the signs of adherent pericardium, paralysis of the left recurrent laryngeal nerve occurred. This was held to have been due to an inclusion of the nerve in the inflammatory pericardial fibrous tissue; on autopsy the nerve was found compressed by fibrous tissue about the arch of the aorta, and it was also pressed on by the left auricle; the nerve was degenerated.

I believe that in subjects under forty years of age both simple and malignant endocarditis are more frequent upon the right side than the current teaching would indicate.

DR. L. HEKTOEN, of Chicago.—Since the reading of my paper on Fragmentation of the Myocardium, the views then expressed have been confirmed by additional study. Segmentation of the myocardium is often accompanied by other degenerative changes, such as fatty and granular changes and vacuolation near the nuclei, which on longitudinal section appear to lie within an empty space: on transverse section the fibrillæ may be absent from the centre. In some instances the nuclei may be very large and may assume anomalous forms. These changes, termed hypertrophic by French authors, are surely degenerative, and may indeed be post mortem productions. With reference to rupture of the aorta, I have recently seen an instance of rupture of an aneurism of the posterior aspect of the ascending arch into the right auricle. In a second case a diffused aneurism of the arch ruptured into the wall of the œsophagus, and dissected downwards into the wall of the stomach into which it ruptured.

Dr. Arthur V. Meigs.—This is a subject in regard to which I feel that I have some right to speak, if an extensive study of the diseased anatomy of the heart and blood-vessels confers such a right. In the first place, I wish to direct attention to the fact that I pointed out some years ago that the circulation of the heart is different from that of other organs. The capillaries actually penetrate the muscular fibres, and there are no minute accompanying veins in the muscular tissue of the heart, the place of such veins being taken by vessels which are similar in structure to capillaries, for they have only one coat. So far as concerns disease, I have stated in one of my publications that the intima is much oftener affected than either of the other coats, and that when the muscularis is diseased, even if thickened, its condition suggests degeneration rather than a true hypertrophy. New vessels are often developed in pathological tissues; for instance, in the valves of the heart, which are naturally avascular, and in cases of adherent pericardium vessels develop in the new tissue deposited between the visceral and the parietal layers of the pericardium. I have never seen such vessels have three coats, but they are more nearly like capillaries, only one coat being distinguishable. In hypertrophy of the heart it is my experience that the tissue is always diseased and degenerated. I have already described these degenerations, and shown how the fibres are often narrower than in the natural condition, and that they are frequently hollowed, or, as it is commonly called, vacuolated. Disease of the fatty covering of the heart is also exceedingly common.

Dr. Coplin.—I regard it as inaccurate to class the serous and the fibrinous pericarditides as distinct forms. I believe the differences are, so to speak, accidental, and due more to surrounding circumstances than to essential differences in the inflammation. In some cases the sera absorb rapidly, in others very slowly and to but slight degree. In some instances the fibrinogenetic substances in the liquor sanguinis and in the exudation are more active than they are in others. The exudation collects slowly in some cases, very rapidly in others. These relations, I hold, are of determining importance in the character of these exudates, whether fibrinous or not.

DR. TYSON.—My experience has not been that pericarditis an nephritis are often associated.

DR. MUSSER.—Does not the fat about other viscera undergo the same pathological changes to which the pericardial fat is subject? It seems to me that fibrous overgrowth and true muscular hypertrophy occur in the same heart. I agree that we are not prepared to say that such hearts are as strong as the healthy heart.

February 24, 1898.

Multiple Outgrowths from the Serous Surface of the Ileum.

FREDERICK A. PACKARD, M. D.

At the autopsy upon the body of a three-year-old colored boy, at the Children's Hospital, the specimens presented were discovered. The child died of tuberculous peritonitis and multiple tyromata of the brain. A full report of the brain lesions will be published elsewhere, with the clinical report of the case.

On opening the abdomen the contents were found matted together and to the abdominal wall, by a greenish yellow plastic material, evidently tuberculous. From this mass the intestines were separated with some difficulty. The small intestine was cleansed by running water, slit up and laid upon the table, when it was noticed that the upper surface of the middle third of the ileum presented what at first sight were supposed to be examples of tricocephalus dispar, but on more careful examination it was found that the exposed upper surface was the serous, and not the mucous, coat. There were found about seventy-five thin white thread-like processes, having an average length of about a centimeter, measuring about a millimeter at the base, and gradually tapering to a quite fine free extremity. Some of the growths were bifid at the extreme tip. On making traction upon them the serous coat of the intestine is elevated, and is apparently continuous with the covering of the outgrowth.

Sections of the growths were kindly made for me by Dr. J. Dutton Steele. They are seen to present quite uniform microscopic appearances. The main material entering into their formation is loose areolar connective tissue within a serous envelope. In each process there is a central canal, running lengthwise, surrounded by a quite dense mass of connective tissue cells, running transversely around the canal. No endothelial lining of the channel can be definitely determined, but the canals are probably blood-vessels. In some of the sections they bifurcate, one branch running into each of the two free ends at the extreme apex. At the base the channel can, in some of the sections, be seen to be apparently a continuation of channels in the subserous connective tissue of the gut. As a rule, they are empty, but in a few of the sections there is at some point a collection of what appears to be the remains of leukocytes.

The wall of the gut shows marked tuberculous inflammation in areas not at all related to the seat of the growths.

In spite of quite thorough search I have been unable to find a description of such lesions. The only explanation of their causation that occurs to me is that they may be the remains of ruptured adhesions. Their appearance, however, decidedly differs from that usually seen as a result of former peritoneal adhesions.

In none of the sections examined is there any evidence of tuberculous inflammation in the growths proper.

DISCUSSION.

DR. SAILER.—I agree that the channels are blood-vessels, but I have not been able to distinguish any elastic layer. The distal end of the vessel presents an abrupt blind ending, which is very difficult to explain.

DR. HUGHES.—Could these be drawn-out peduncles of tuberculous growths which had in some way disappeared? Both the distribution and appearance are however against such a view. They are obviously not neoplasmic, perhaps they might be reversionary structures.

Dr. Packard.—I suggested that they might be the remains of adhesions, but it cannot be conceived why the vessels were not closed and obliterated by process of thrombosis and organization, instead of remaining patulous to the blind extremity. The appearance of the growths differ decidedly from that of any other instance of ruptured peritoneal adhesions seen by me.

February 10, 1898.

A Tubal Gestation-Sac with Unruptured Membranes.

H. L. WILLIAMS, M. D.

The specimen here presented was removed by Dr. B. C. Hirst, from a patient in the University Hospital, and is especially interesting in that it occurred in a tube the seat of a large pyosalpinx; that it is at a very early stage of development, and that the membranes were still unruptured at the time of its removal. The history and symptoms were not such that a diagnosis of gestation could be made prior to operation. The patient presented the typical symptoms of chronic tubo-ovarian disease, and on examination a diagnosis of salpingitis was easily established. At the operation, an immense pyosalpinx was removed, from the left side, in which was found the gestation sac with membranes unruptured.

February 10, 1898.

Carcinoma of the Common Bile-Duct and Head of Pancreas.

W. E. HUGHES, M. D.

The patient was a man *aet* 80 years, who was admitted to the Philadelphia Hospital January 19, 1897. At this time he complained of little but senile debility. He had had shortness of breath and dropsy forty years before, but had been cured. With this and some minor exceptions, there had been no illness all his life. He did not use alcohol, and gave no venereal history.

On admission he had a cough and slight dyspnœa, due to pulmonary emphysema. The veins of the legs were varicose. There was a large reducible inguinal hernia, which had been present for six years. The liver and spleen were normal and his digestion good. July 16 he complained of a little pain over the epigastrium and region of the liver and around the waist, and was slightly jaundiced. For some time previous to this he had been coughing, and had been complaining of weakness. The jaundice steadily increased, becoming eventually very marked. The stomach was often irritable, the bowels constipated and the stools devoid of bile. Through these days there was no enlargement of the liver, spleen, gall-bladder or abdominal veins. His strength steadily failed and his weight declined. On October 3, there having been no examinations made for some weeks, there was noted a palpable ovoid cystic mass in the abdomen, the apex of which extended to the edge of the liver, the base lying obliquely across the abdomen to 2½ inches beyond the median line. A line drawn from the umbilicus to the anterior superior spine bisected it. At this point it was 2½ inches wide. The patient was much emaciated, profoundly anæmic and deeply jaundiced. The urine had varied in specific gravity from 1012 to 1030, contained occasionally a trace of albumen, but never sugar, and was uniformly heavily loaded with bile. The bowel movements contained no bile and no exceptionally large amount of free fat. He died October 20.

On autopsy the gall-bladder contained 20 oz. of rather pale bile, but no stones. From the bile-ducts, which were extremely dilated, could be pressed out about 25 oz. more bile. The liver presented a peculiar appearance, owing to the immensely dilated ducts. Its substance was deeply bile-stained and somewhat fibroid. The bile contained, on microscopic examination, granular debris and cholesterin. The mucous membrane of the bile-ducts was normal. The biliary papilla projected as a blunt mass far into the duodenum. This mass was composed of the thickened wall of the end of the common bile-duct, which, for ½ inch from its termination, was composed of dense, firm, whitish tissue. The lumen of the duct was completely obliterated, but down to the point of obliteration the mucous membrane did not seem to be affected. The pancreas showed no gross lesions. Its ducts were not dilated. There was no enlargement of any of the abdominal lymph glands. The lungs were extensively emphysematous. None of the other organs were remarkable. Microscopic examination of the thickened wall of the common bile-duct, made by Dr. Riesman, showed it to be the seat of glandular carcinoma. This carcinoma had begun to infiltrate the head of the pancreas.

The cancerous process had been probably set up in a cicatricial condition of the end of the common duct, but there was nothing in the clinical history of the case to throw any light on the causation of this cicatrix.

Clinically, the case is interesting from the point of view of the diagnosis of cancer of the pancreas. This diagnosis was made during life, and was based on the occurrence of jaundice and an enlarged gall-bladder, without any decided change in the liver or obvious reason for their production. These diagnostic points are unquestionably of great value in differentiating between cancer of the head of the pancreas on the one hand, and gall stone or primary cancer of the gall-bladder on the other. While the diagnosis in this case was not absolutely correct, it must be pointed out that it is altogether impossible to make a diagnosis between cancer of the head of the pancreas and the much rarer condition, primary cancer of the common bile-duct.

DISCUSSION.

DR. PACKARD.—I recall a similar case. The patient had emaciation without gastric symptoms, jaundice was present, but there was no hepatic enlargement, there was a tumor at the site of the gall-bladder. The necropsy revealed a primary carcinoma of the head of the pancreas and a secondary circular infiltration of the gall-bladder. I noticed the sign mentioned by Bard and Pic, viz:—that continued palpation served to make the tumor of the gall-bladder more apparent, it seems in some way to bring it out more distinctly under the hand.

DR. RIESMAN.—I have recently studied a case of a similar kind. The patient, a man of fifty years, was markedly cachectic and jaundiced; he did not complain of gastric symptoms, the liver was not enlarged, there was no tumor palpable, glycosuria and steatorrhœa were not present. At the autopsy a primary carcinoma was found in the head of the pancreas, the gall-bladder was not involved, nor was it materially enlarged. The tumor, which I shall later exhibit, was a squamous carcinoma; there were metastases in the liver of the same nature as the primary growth.

DR. SCHREINER.—I recall a case which presented marked cachexia, hepatic enlargement with jaundice, and a tumor in the region of the gall-bladder. On post-mortem examination the jaundice was seen to have been caused by pressure upon the common duct by the gall-bladder, while the tumor was an ecchinocchus cyst of the liver. *February 10, 1898.*

Description of New Apparatus.

W. M. L. COPLIN, M. D.

NEW PARAFFIN OVEN FOR CLASS-WORK.

This apparatus consists of a cylinder 49 cm. in diameter, 20 cm. in height, outside measurements. The entire apparatus is made of copper; the bottom is extra heavy; the top consists of a sheet of copper which contains 99 perforations into which are brazed, water tight, metallic cartridge shells 4 cm. deep and having a diameter of 2.5 cm. Into these are placed the cylinders in which the paraffin infiltration is done. These cylinders are 6.5 cm. high, 2.2 cm. in

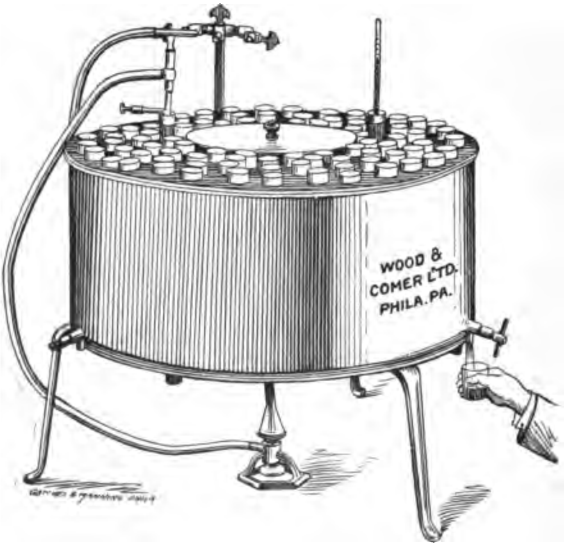

FIG. 1. PARAFFIN OVEN.

Thermometer and thermoregulator are shown in position. On the left, near the leg, is shown one of the cocks for drawing off the paraffin from the central reservoir. On the right is shown the method of filling and embedding the cylinders from the central reservoir. A third cock is on the opposite side.

diameter. There are also perforations for filling the oven, for the water gauge, and perforations for the thermometer and thermoregulator, the last four perforations communicate with the water chamber. In the centre of the cylinder extending 1 cm. above the top is a reservoir 19 cm. in diameter and 14.5 cm. deep and having a capacity of 1200 c.c. Running from the bottom of this reservoir, which is concave, are three tubes which extend outward, as radii, through the water chamber and through the outer wall at which point they are directed downward in such a way as to drain the paraffin from the reservoir. Each of these is controlled by a specially constructed cock which closes the tube inside of the water reservoir, thereby preventing the solidification of the paraffin in the tube outside of the water reservoir. This part of the apparatus rests upon a three-legged stand made of cast iron and having for its cover a sheet of copper which prevents the burning of the copper bottom of the cylindrical tank above described.

The advantages of the apparatus are that it enables forty-nine students to carry out paraffin infiltration, each student having two containers of paraffin. The central reservoir affords the student a supply of paraffin for embedding. Two paraffin cylinders are assigned to each student and are his personal property during the time he is working in the laboratories. By the use of this oven we have been able to have every student infiltrate practically all his own material.

A Simple Arrangement for Suspending the Stewart Cover-Glass Forceps in the Author's Dish or in the Stender Dish.

This is made from a piece of flexible copper wire 20 cm. long. One end of the wire is passed through the round opening intended for the thumb, on the top of the Stewart forceps and down through the right end of the sigmoid at the bottom of the forceps. It is then wrapped around this sigmoid and carried to the right so as to project 3 cm. from the right end of the sigmoid. The opposite end is now turned and passed through the upper round opening and down through the left end of the sigmoid, and bent around the sigmoid, as already described for the right side, and carried 3 cm. to the left. This device is now so arranged that the two ends running laterally from the sigmoid rest on the top of the dish, and the loop made above the round turn in the top of the Stewart forceps projects sufficiently far to rest upon the top of the dish, thereby giving three points of support. This arrangement is constructed for staining blood, sputum, bacteria, etc., and may be used with the cover-glass method of albumen fixation of sections.

A Box for Carrying Blood Films.

This consists of a rectangular box with detachable top and bottom. It is 2 cm. wide, 6.3 cm. long and 3.5 cm. high. On the inner wall of the two sides are arranged clips which grasp the cover-glasses, holding two cover-glasses, back to back, in each clip. The box is at present made to carry any cover-glass which has a diameter of 2 cm. and which has a length not exceeding 3 cm. On one side of the box opposite the cover-glasses are a series of numbers running from one to twelve and corresponding to the cover-glasses in the clips on the inside. The top and bottom of the box which are open are closed by means of what might be called two lids. Upon one of these there is an arrangement for carrying a small pair of forceps. The box is put together with brazing solder so that after the films have been collected the whole box can be placed in the hot air oven for fixing, or, after removal of the lids, will fit into a Stender dish in which any fixing solution may be kept.

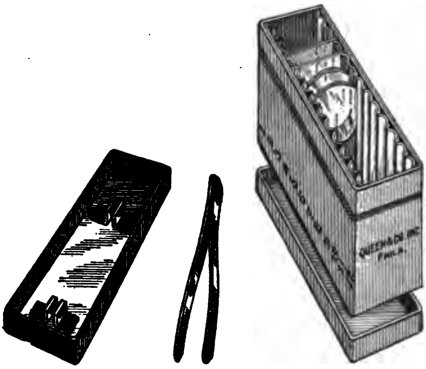

Fig. 2. Box for Carrying Films.

On the left is shown the lid with the holder for grasping the forceps. On the right is shown the box with four round cover-glasses and four square cover-glasses in position. The movable bottom is slipped off, a short distance, to show how it separates. On the left side of the box are seen the figures for the identification of the corresponding cover-glasses on the inside.

The improved box, which unfortunately I have not been able to exhibit this evening, will have the inside measurements as follows: approximately 75 mm. square, 35 mm. high. One side of the box will have clips arranged as in the present box. Opposite these will be a movable side which can be adapted to carry any size cover-glass, giving space for thirty cover-glasses. The remainder of the box will be so constructed as to hold slides, easily accommodating a half dozen slides with the central division to hold forceps, immersion oil, paraffin for sealing the cover-glass to the slide, etc. This arrangement will afford carrying capacity for six to ten mounts and thirty cover-glasses, forceps, oil and cement. There will be a similar arrangement for numbering the cover-glasses as in the present box, and the slides will be identifiable by letters.

February 10, 1898.

Acute Epiphysitis and Purulent Pericarditis Complicating Typhoid Fever.

FROM THE SERVICE OF DR. MARSHALL, ST. JOSEPH'S HOSPITAL.

H. SMITH, M. D.

The patient was a white girl aged eleven years. The family history was negative. She was a delicate child, but had never had any serious illness. The week following Christmas she complained of pains in the limbs and head, languor and general malaise. With an aggravation of these symptoms, and attacks of vomiting, she went to bed January 4th, and was admitted to the hospital four days later.

Examination on admission showed the typical typhoid facies, abdominal tenderness, and a few rose-colored spots. The heart and lungs were normal. A mild delirium was present. She complained bitterly of pains in the right thigh and knee. She gave a history of having fallen about December 28th, injuring the knee, but the pain did not become acute until January 6th. Examination of the thigh showed no perceptible swelling or redness, but an exquisite tenderness over the whole posterior surface, especially back of and above the knee.

On January 11th, she complained of præcordial pain, and on the following day a friction sound was distinctly heard over the base of the heart. The pulse increased to 140 and the respirations to 40 per minute. On January 14th, an effusion appeared. The thigh now became somewhat swollen, œdematous and slightly red. The leg was constantly flexed on the thigh, and the weight of the bed-covers caused much pain. No characteristic bone pains were present. The typhoid fever was apparently running a favorable, although a severe course, with much delirium, rapid pulse and respirations; but the leg and heart conditions steadily grew worse. At no time were there chills or sweats, although the temperature assumed a hectic character. Neither were there any pressure symptoms from the large pericardial effusion. Death occurred on February 1st.

Necropsy: External examination showed a bulging of the præcordia and swelling of the right thigh. Upon removal of the sternum the pericardial sac bulged forward and was tense, extending from just beyond the right sternal border to the anterior axillary line on the left and to the second rib above. It contained, under pressure, an odorless fluid of creamy color and consistency. The parietal pericardium was covered with a smooth, white layer of lymph, several millimeters in thickness, which stripped off readily, exposing a hyperæmic and ecchymotic surface. The visceral pericardium was entirely covered with white nodules of contracted lymph, the "cor villosum." The endocardium was normal. The left lung showed a recent fibrinous pleurisy. In both lungs were found irregularly distributed hemorrhagic infarcts, small abscesses, and thrombi in the pulmonary veins. The stomach was greatly dilated, completely hiding the transverse colon. Patches of petechial hemorrhage were present. There was no pyloric obstruction. The spleen was *not* enlarged. The liver was displaced downward. The right kidney contained a hemorrhagic infarct. The bowel, close to the ileo-cæcal junction, contained ten or twelve healing typhoid ulcers; also patches of petechial hemorrhage. The mesenteric glands were much enlarged. The right thigh, posteriorly, was one large abscess cavity. The muscular tissue was dissected from the bone. The periosteum had been destroyed, leaving a smooth, white surface, with no signs of necrosis. Pus was found in the knee-joint. The medulla of the bone was a dark brownish red, and contained some pus.

Cultures taken from the pericardial effusion, the abscess in the thigh, the pus in the knee-joint, and the medulla of the femur showed the staphylococcus pyogenes albus. Cultures made from the spleen were negative.

The interesting feature of this case is the apparently simultaneous development of typhoid fever and an acute epiphysitis, which latter disease was quickly followed by an extensive osteo-periostitis, and later complicated by an acute purulent pericarditis. The diagnosis of typhoid fever was based on the following facts: the symptoms were typical, the eruption was present, the Widal reaction was repeatedly obtained with as great as a 1 to 80 dilution, and post-mortem examination showed healing ulcers in the bowel. The femoral condition apparently started as an acute epiphysitis following an injury, the periosteum and adjacent soft tissues quickly becoming involved, and the pericarditis developed early as a complication.

DISCUSSION.

DR. JOHN H. JOPSON.—I reported before the Pædiatric Society last year a case of suppurative osteomyelitis in a child in which the post-mortem findings included what were apparently typhoid ulcers in the intestine. It is difficult to diagnose typhoid fever in children from intestinal ulcers alone, but in the case presented to-night the diagnosis seems to have been amply confirmed. Klemm has produced a liquefaction of the bone marrow in dogs by injections of typhoid bacilli, and by the subsequent injection of staphylococci has produced a typical suppurative osteomyelitis. Unless we could prove the pre-existence of the bone lesions in this case, it would be more justifiable to view it as an osteomyelitis from a secondary infection due to implantation of the staphylococcus on a bone marrow weakened by the invasion of the typhoid bacillus, rather than as a mere accidental association of the two diseases.

DR. SAILER.—The cocci in the smeared preparations of the pus in this case refused Gram's stain, but the cultures showed them to be the staph. pyog. albus. I believe that the suppuration in the bone was staphylococcic, but the primary lesion there may have been due to the typhoid bacillus. In regard to the possibility of a secondary infection with the staphylococcus, in the course of the typhoid fever, I should be inclined to exclude it in the present case, chiefly on account of the history, in which it is distinctly stated that the symptoms in the thigh appeared at least as early as those of the abdominal infection. Mixed infection is the rule in many of these cases. Flexner, in one case, found four varieties of parasites, two bacteria, and two protozoa. *February 10, 1898.*

A Case of Congenital Enteroptosis.

JOSEPH SAILER, M.D.

The patient (a white male) was brought to the hospital suffering from uræmia and in a delirious condition. His occupation had been that of groom in a stable, where he apparently had had rather severe work. Nothing is known as to his habits or previous health. He died soon after admission. Inspection showed the body of a well-nourished man between forty and fifty years of age, moderately muscular, with double pes planis. There were no other external marks. Upon opening the abdomen the transverse colon was observed occupying the lower part of the abdominal cavity, resting just above the symphysis pubis; to either side it curved upward and backward; but the hepatic flexure was entirely clear of the lower border of the liver, and the splenic flexure lay just above the left kidney, from which place the descending colon passed in an almost straight line to the rectum, the only indication of the sigmoid flexure being a slight deviation to the right just as it crossed the prominence of the sacrum. The ascending colon was short and the appendix and cæcum were in their normal situation and bound to the abdominal wall by rather firm adhesions. The liver was greatly enlarged, extending three inches below the costal border in the right nipple line, and four inches below the xiphoid cartilage; and the left lobe reached as far as the left anterior axillary line. When removed it was found to be in a state of fatty infiltration, and weighed seven pounds three ounces. The small intestine lay partly above and partly below the transverse colon, the lower portion filling the upper part of the cavity of the pelvis. A single loop from the ileum descended as far as the right internal inguinal ring, but did not pass through it. The pyloric end of the stomach appeared just below the lower border of the xiphoid cartilage. The greater curvature was about three inches below the border of the ribs, the long diameter of the stomach being placed at an angle of about forty-five degrees. The stomach presented the picture of chronic hypertrophic gastritis, probably due to alcoholism. The spleen was in its normal position, weighed nine ounces, and beneath it there was a small supernumerary spleen. Both kidneys were in their normal positions, firmly attached to the posterior abdominal wall, and imbedded in a large mass of perirenal fat. They showed extreme congestion, moderate interstitial nephritis, and fatty infiltration of the pelvis. The bladder and intestines were negative. The upper portion of the abdominal cavity was approximately normal. The diaphragm reached to the fourth interspace on the right, and to the fifth interspace on the left side. The pelvis was unusually capacious. I have rarely, even in women, observed such a large cavity. It was almost circular in form, and the sacrum projected backward almost perpendicularly to the spinal column. It easily contained the distended bladder and about half of the small intestines. Unfortunately, in the absence of a pelvimeter, it was impossible to take any measurements. The tenth rib was not tested for Stiller's sign, the floating tenth rib; but it was attached by cartilage to the ninth rib.

Curschmann[1] a few years ago described some of the anomalies of the large intestines that he had observed in autopsies. Among these were shortening of the ascending colon, with a high position of the lower portion, and the absence of the hepatic and splenic flexures. At his instigation, Schulze[2] examined the work of Morgagni for a description of similar changes, and found nineteen cases recorded, with a general reference to others that he had observed. Of these nineteen, the majority showed simple downward displacement of the transverse colon, its lowest portion lying usually beneath the navel. This condition must have been associated with increase of the total length of the colon, because the flexures were usually in their normal positions. In one of these cases, the arrangement, however, was somewhat different. The colon did not pass from the right side directly to the left, but, at the point where it reached the fundus of the stomach, turned downward through the abdominal cavity without bending, and united with the rectum. The present case is, I think, particularly interesting on account of the typically congenital nature of the arrangement. There can be no question of the action of a corset or pregnancy. Neither could the enlargement of the liver account for the condition, although, from a sentence translated from Morgagni, it seems not entirely unlikely that he sometimes observed hepatic enlargement associated with anomalous positions of the colon. Langerhans[3] lays considerable stress upon the inverted conical shape of the abdomen, and believes that the intestines are supported without pressure in the cavity, and therefore cannot fall of their own weight downward. This explains the absence of the entrance of air through the stomach sound. He recognizes five causes for enteroptosis: (1) weakness of the supporting muscles, that is, the abdominal wall; (2) an hereditary enteroptotic predisposition; (3) corsets; (4) chlorosis; (5) nervous dyspepsia. I am inclined to look upon the condition in my case as due to the alteration in the shape of the abdominal cavity. This can fairly be said to be cylindrical, and, as by the simple action of gravity, the lower portion of the abdominal contents are subject to more pressure than those situated higher, they would be naturally forced downward if there were space to contain them. In the present instance, this space existed in the abnormally enlarged pelvic cavity, and at the same time there must have been a congenital lack of longitudinal development in the colon itself, and unusual elongation of the mesentery and mesocolon. The absence of a distinct sigmoid flexure is apparently exceedingly rare. It is not mentioned by Orth, Ziegler, nor Birch-Hirschfeld; Curschmann fails to record a case, and Morgagni only one. There is no reason to suppose that in the present instance any symptoms existed during life. Nutrition was certainly not interfered with, for the layer of subcutaneous fat was quite thick. Unfortunately it is impossible to find out whether there was chronic constipation, the absence of fecal masses in the intestines being probably due to the effect of purgative medicines. The presence of two anomalous conditions, besides the intestinal and pelvic changes, viz., double flat foot and the supernumerary spleen, are interesting in view of the frequent association of congenital defects.

(1) Curschmann.—Deutsches Archiv. für Klinische Medicin, Vol. liii, 1. (2) Schulze.—Ibid. Vol. lix, 5 and 6. (3) Langerhans.—Archiv. für Verdauungskrankheiten, Vol. iii, 3.

DISCUSSION.

DR. COPLIN.—Ptosis of the colon is common in the inmates of the insane department of the Philadelphia Hospital, due probably to their habits of eating and to constipation.

DR. EDSALL.—The greater disposition of the female sex to enteroptosis is usually ascribed to constriction, lax abdominal walls and other acquired causes. Dr. Sailer's case suggests that the female pelvis from its roominess, affords a natural predisposition to descend of the viscera if their attachments or support from above be loosened.

Dr. Swan recalled a case of ptosis in which the colon was on a line with the superior iliac spinal processes; both flexures were present.

Dr. Daland recalled an instance in which a descended transverse colon, dilated with gas, had simulated gastrectasis.

Dr. Sailer.—When Assistant Pathologist at the Philadelphia Hospital, I recorded a number of instances of anomalous arrangements of the abdominal contents. The most frequent variation was elongation and descent of the transverse colon. There was frequently an abnormally large loop from the sigmoid flexure, and in one instance this extended diagonally upwards as far as the lower border of the liver. None of the cases, however, resembled the one I present this evening.

February 10, 1898.

Typhoid Fever, with Irregular Lesions.

D. L. EDSALL, M. D.

This case is presented as typhoid fever, because its clinical course and the post-mortem lesions correspond more closely to that disease than to any other. The patient was a stevedore, thirty-eight years old, whose history, in brief, was that he had begun to feel ill about four weeks before I saw him; had headache, epistaxis, diarrhœa, and extreme malaise. He kept at his work, however, until a week before I saw him, when he became so prostrated that he was obliged to go to bed. When seen, he was in a stuporous condition, could be aroused with difficulty, but was rational and presented no evident abnormal physical signs, though he had a temperature usually about one and a half degrees above normal. His abdomen was flat; his spleen not enlarged; he had constipation; his lungs were clear and his heart-sounds weak, but clear. Beyond his stupor there were no symptoms pointing to disease of the brain or its membranes. The urine contained but a trace of albumen, but nothing else abnormal. Four days after I first saw him he sat up in bed, and in a moment fell back, dead.

The post-mortem examination showed, in the abdomen, old adhesive peritonitis about the spleen, liver, and pancreas; the intestinal coils were almost entirely free. The liver was somewhat congested and slightly enlarged; the spleen was enlarged perhaps one-quarter beyond the normal, and its capsule was wrinkled as if more enlargement had been recently present. The kidneys showed slight cloudy swelling, but no marked changes. The supra-renal capsules were firmly entangled in adhesions, and their centre appeared somewhat caseous. The stomach was normal, the large intestine was much congested in the upper part, but there was no ulceration and no marked inflammation. There were ulcers in the small intestine, but of these the nearest to the ilio-cæcal valve was about four and a half feet distant, and the ulcers were of very unusual appearance. The three or four nearest the valve looked like small typhoidal ulcers in the healing stage, their edges were clean, and their bases free from slough and cicatrizing. Beyond these there were a number of larger and much more irregular ulcers. One of them encircled the entire bowel, and was about two inches broad. Another was nearly as broad as this, and it, too, encircled the bowel. The others, though smaller, were of very irregular form. The edges of all these were clean, not overhanging, their bases granular, and all were apparently healing. The edges of the large ulcers were thickened somewhat, of quite regular outline without indentations, while the smaller ulcerations were of very irregular shape, not at all like typhoidal ulcers, their long diameters not bearing any relation to the course of the bowel. The bases felt rough and firm and were of considerable thickness; the loss of tissue evidently extending through the mucusa and perhaps the sub-mucusa alone. The largest ulcer only had clinging to its base a small slough. The serous coat of the intestine looked, over these ulcers, as well as elsewhere, entirely normal, excepting for some congestion. Beside the ulceration, the small intestine showed a great deal of congestion, especially in its lower part, and there were some slight cicatrices, as if from small healed ulcers. The mesenteric glands were moderately swollen. The heart had stopped in firm systole, and was contracted in a peculiar form, so that its apex looked extremely pointed. The ventricles and auricles contained current jelly clots, the valves were normal; there was a quite firm thrombus in the pulmonary artery, as large as an index finger, and this extended for five inches into each main branch. The lungs were large, deeply congested, and showed an extreme degree of œdema, so that when cut, serum ran out in small streams. There was no consolidation in either lung. Examination of the brain and spinal cord could not be undertaken. The Widal test had been made during life by Dr. Kneass, and gave a very doubtful reaction. It was done by the dry method, and before an opportunity was offered to make an accurate dilution death had occurred.

I examined the spleen microscopically without finding any typhoid bacilli, although a number of sectious were gone over. There was a considerable degree of round-cell infiltration in the lymphoid follicles, the vessels somewhat widened. The kidneys showed microscopically a moderate cloudy swelling and a slight desquamation of the cells, with slight round-cell infiltration. Sections from the heart showed round-cell infiltration, the nuclei stained badly and the striations of the muscular fibres were indistinct and in many cells lost. There was no fragmentation of the fibres. Sections from the intestinal ulcers showed an entire loss of the mucous coat, the surface being formed of fibrous tissue of new formation. Beneath this, in the submucosa and muscularis, were a small number of round cells, while upon the surface and that of the nearby mucous membrane there was a layer of mucus entangling a large number of micrococci and short, plump bacilli.

The lesions evidently do not correspond to the typical lesions of typhoid fever, and the imperfect Widal test throws much more doubt upon such a diagnosis. I do not think that the absence of bacilli from the spleen is of much importance, since the man had certainly about recovered from the disease, if it were typhoid fever. It can scarcely be considered as a dysentery, because of the situation of the ulcers, their healthy character, and the clinical manifestations; nor were the ulcers tuberculous, since they have, upon microscopic examination, absolutely no appearance of tuberculosis, nor was there even macroscopic evidence of it. The clean surfaces of the ulcers and their appearance of cicatrization, would seem sufficient evidence that they were not the necrotic ulcers that occur in meningitis, diabetes, and other affections; nor were they from coprostasis for the same reason, and because no marked constipation existed. The condition of the kidneys was scarcely grave enough to have caused death by uræmia, and the suddenly fatal termination seems best explained by the large pulmonary thrombus and the œdema of the lungs.

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TABLE OF CONTENTS.

WADSWORTH, The Pathology of Color Perception —**WILLIAMS**, A Case of Extra-uterine Pregnancy.—**SCOTT**, Specimens from a Case of Pernicious Anemia.—**MCFARLAND**. A Case of Aneurysm of the Aorta with Rupture into the Right Pleural Cavity.—**WILLIAMS**, A Dermoid Cyst Obstructing Labor and Requiring Cæsarian Section.—**HUGHES**, Rupture of Kidney and Spleen. Heart and Lungs from a Bicycle Rider, Pericarditis in Bright's Disease.—**TAYLOR**, Aneurysm of the Arch of the Aorta. Hour-Glass Stomach. Marked Fibroid Myocarditis, with Extensive Valvular Contraction. Sarcoma Presenting Many Mastcells.—**GAYLORD**, Hepatic Embolus in the Lung. Illustrations of Cardiac Lesions. Card Specimens.

The Pathology of Color Perception.

WILLIAM S. WADSWORTH, M. D.

The few observations I have to present to you to-night are part of the result of a long study of the phenomena of color, extending over a period of seven years, and including work done in three universities and observations made in many places.

While we are limited in our present considerations to things pathological, it is essential that we should have a clear understanding of the fundamental processes of color perception, in order that we may know when we are on physiological and when on pathological grounds.

Color perception is the outcome of a rather complicated action of many functions and these functions acting in harmony.

In the past it has been usual to attribute all lesions of color perception to defects of the retina, these defects being not infrequently of a highly imaginary nature. No one would for a moment think of denying that the retina plays a most essential part in color perception. But to one at all familiar with the physiology of color the disregard of the mental functions which is so common in prevalent color theories is more absurd than an equal overlooking of the retina. To stop at the retina and disregard the great system that lies behind and of which the retina is indeed but a small part, is so mistaken that we wonder how it can be done by intelligent physiologists.

Color perception is the reaction of the whole system to the colored light in the eye.—Sweeping as this statement may seem, and open to objection as it seems to be, it will bear the intelligent investigation and criticism of mature study.

This reaction depends on three factors: (1) the condition of the eye; (2) the condition of the mental functions; (3) the amount and nature of the stimulus.

The conditions of the eye that give rise to defective color perception are many, familiar examples are hemorrhages in the eye, scotomata, yellowing of the humors, etc.; but it is not my purpose to discuss the ophthalmological side of defective color perception, because my experience leads me to believe that wherever color perception is seriously disordered the eye symptoms are secondary to the general condition.

The condition of the mental functions is of the greatest importance, this is naturally to a large degree dependent on the general health, but aside from the direct bodily influences we find that the mental states form very important factors, not only in the normal perception of color, but also in the pathogenesis of abnormal color perception. The separate mental functions have peculiar relations to the process, certain of them being most intimately and essentially associated with it. The attention is perhaps the best example; diseases of the attention produce very marked and usually characteristic lesions of the color sense, rendering the results of the activity of the functions that are concerned in color perceptions uncertain or at times wholly wrong. There seems to exist a very intricate relation between the attention and the color sense, for we find functional derangements affecting a group of functions in such a manner that we cannot regard the association as accidental. Thus we know that emotional excesses cause debility and disease of the attention, and I have very good reason to believe that these same emotional excesses have a special effect upon the color sense through its specific functions.

The mental functions of the inner or more truly psychological set, those that have to do with the co-ordination of the other functions of the mind, those governing the flow of ideas and the awakening of memories through the association processes, those that have to do with the moral nature, and functions that give rise by their derangement to all such diseases as stuttering, kleptomania, nymphomania, etc., may be involved in the disturbances, and when they are the color perception is not infrequently affected in a way that is surprising to those unacquainted with mental phenomena.

Naturally the great majority of men are comparatively healthy in color function, but from an absolute standpoint very few are perfectly normal. By the crude wool tests of Holmgren, about 4 per cent of all males are color blind, by more accurate methods a much larger percentage would be found to have lesions of the color sense. As only a small number of the 4 per cent are totally color blind, the mass being merely color-defective, those detected by finer methods would have correspondingly light lesions.

The earlier tests, which were made with almost any colored substance that came to hand, occasionally yielded very accurate results, but the uncertainty of the tests, from a lack of uniformity, rendered a careful analysis almost impossible. Holmgren and his followers were able to detect defective color vision, but that they continued to follow Helmholz was significant.

Hering and others have attempted to formulate more philosophical theories of color vision, yet few to-day seem to realize that the problem of color perception is a matter, not of physics, nor yet of chemistry, but that it is a problem of biology, and that the vital conditions of the protoplasm of the cells of the nervous system, from the retina to the ultimate perception centres, is the essential and only essential thing, and that mechanical phenomena are but the indications that the subtle protoplasmic functions are modified.

The pathogenesis of defective color perception is like the pathogenesis of other mental and nervous disorders, it obeys the same laws, and a study of this one field of pathogenesis reveals fundamental laws that govern mental growth, activity and degeneration. Defective color perception may arise from two sides, from the outside by injury to or some local affection of the eye, especially when the retina is involved, or it may arise, and does, undoubtedly, in the majority of cases, from an internal condition of the nervous system.

Color blindness is so markedly a functional disease, even in its most accentuated forms, that it seems unprofitable at the present time to ask for anatomical explanations. We can, however, study the progress of color degeneration, and by observing the types of mental and bodily activity with which it is associated we can learn some very deep truths of pathogenesis.

Looking at this card which I hold you all agree that it is red; now, looking constantly at it, some of you will feel that it is not so bright as it was at first; if you look at this pencil, which I hold at a distance of about a foot from the red card, some of you will notice that the red color fades more rapidly. Now the sending of that message of red from the retina to the mind has used up energy and left certain cells in the nervous system, to a degree, exhausted; and furthermore, the reception of that message in certain cells of the central nervous system has to a degree exhausted those cells; the degree of exhaustion will depend on their native strength, on the condition they were in before stimulation and on the amount of stimulation.

I now hold before you a second red card. It is only one-half as red as was the first card; to some it will fade very soon. When the cells become exhausted they respond less readily to a stimulus, and, although the weaker red exhausts them more slowly, they cease to respond to the weaker stimulus. Now suppose that the cells, either in the retina or in the brain, have been reduced by any process to a condition of relative insensitiveness, then a weak red may from the first appear as a gray; the process, however, is more complex. Look on this red card, and as I drop it the place where it was appears green; but the greenness of the after-image differs with different states of the cells, which states cannot be expressed in terms of simple exhaustion; they depend on vital conditions that baffle our keenest analysis, the best we can do is to find the relation that exists between certain conditions of the color sense and certain vital functions.

If I find a hitherto healthy man, after a prolonged indulgence in one sort of emotion, developing a strong distaste for a certain color, I note a phenomenon that is suggestive of pathogenesis. If we see an over-emotional person condemning scarlet we find food for thought, and if we find the discoveror of color blindness belonging to a religious order that debars red as unholy, we suspect that the emotions have a very close relation to the action of those cells which act in the process of color perception; and, going a step further, I find ample confirmation of the suggested relation in all the arts which appeal to the emotions through the eye. The emotions have a peculiarly strong influence on the vital conditions of the cells concerned in color perception, and emotional excesses have a marked influence on color vision; but here again the problem becomes complex, because it appears to depend on a morbid manifestation of the emotion, more than on the gross type of the emotion.

We all noticed these things in a half unconscious way when we were children; when we looked into the eyes of people who came near us, we interpreted those signs that lay written in the cells of the iris, and in the skin and muscles of the face, into emotions that we liked or disliked, and we were very keen in those perceptions. Now as adults we have to learn slowly what we grasped at a glance as children. We now require tests to prove such conditions to our dull perceptions, and I will ask you for a moment to examine the color test, which, with its imperfections, is nevertheless the most usable test that I have yet seen.

The use of the Bradley papers makes the test possible, and I wish to pay a proper tribute to our American paper maker, who has given to the world the first approach to a scientific color standard. These charts are illustrations of cases and were prepared for the third International Congress for Psychology, held in Munich, in the summer of 1896. They illustrate points that have been already spoken of, especially those of the relation of the various mental functions to color defect.

The tests were reduced to a provisional physiological basis by a series of fifty tests, which are also tabulated on the charts.

I will also ask you to compare them with these tables, which show the tabulation of over two thousand readings made by different types of color defective persons before I had elaborated my present test.

The test consists of a set of 100 slips of colored paper 2 x 1 inches. Each slip has an estimated value, and the whole 100 slips give a graded system of spectrum and blended colors. The ten test slips are of the six spectrum colors and four confusion colors. Each slip of the hundred belongs with one and with only one of the test-slips, and the way the person being tested distributes these 100 slips among the ten boxes gives a very clear basis for judgment regarding the color perception of that person.

Directions for Use of Test.

Select a window with a good north light. Place a table about two metres away, in front of window. Place a sheet of white paper on the table and arrange the slips and boxes on paper thus:

* * * * * Boxes,
* * * * * Slips,
* * * * * Slips,
* * * * * Boxes.

Be sure there are no shadows on the table or on the slips. (Strongly colored objects near the person being tested may disturb the test.) Have the person comfortably seated. Have the colored slips well shuffled. The person to be tested must understand exactly what he is to do. To fill the record blank write the number of the box in the space opposite the printed number, which corresponds with that on the slip.

The person using the test should keep all materials used in the tests from exposure to direct sunlight. If possible, all tests should be made at the same time of the day. See that the person being tested sorts the slips by direct comparison with test-slips. Note any comments made. When possible, make notes on the family traits and personal peculiarities, specially regarding nervous, religious, sexual, and other emotional habits; also make notes about work, rest, stimulants, etc. Note the physical peculiarities, specially color of eyes, hair, skin; condition of teeth and digestion, reflexes, and the other sense organs. In these, as in all physiological tests, the greatest care must be taken to avoid disturbing conditions, specially such as affect the eye directly.

The person to be tested should take each slip separately and match with one of the test-slips; it must be matched by the color and not by the shade. Each slip belongs with one of the test-slips. The slip should be placed in the box belonging to the selected test-slip. A slip must not be touched after being placed in the box, the slips being taken in the order in which they are found and placed as rapidly as possible. No attention should be paid to slips already placed. One must not try to harmonize the tests. If in doubt, one should rest a moment and then place the slip where it seems to belong. Never reason about color theories, but sort the slips by the sensations they give.

DISCUSSION.

DR. A. C. ABBOTT.—In this connection I would refer to an interesting case in a friend of mine. He is something of an artist, paints extremely well in colors, despite the fact that he is color-blind. Has Dr. Wadsworth any knowledge of similar cases? Has he also made any studies upon the color perception of children —particularly upon the preference of children for particular colors?

DR. SPILLER.—The exhaustion of nerve cells after activity, to which Dr. Wadsworth has referred in speaking of color perception, is well known. We see it manifested in many ways. Not infrequently we find an ankle donus, which is quite evident on the first attempt to elicit it, but soon disappears. It is hardly necessary to refer to the investigations of Hodge and one or two others. I have been hoping to hear something from Dr. Wadsworth in regard to color perception in hysterical patients, for it is among these that we find some of the most remarkable abnormal conditions, which must be the result of laws, even though they may be unknown to us. I have, for example, a patient who can distinguish red and blue and can read with her left eye when the right is closed, but is unable to distinguish color or read when the right eye alone or both together are opened. She has also reversal of the color fields.

DR. WADSWORTH.—Several color-blind painters have been reported. Dr. Jeffries told me of one who always had his colors arranged for him before he commenced to paint. There seems to be a degree of compensation for the loss of color-sense by an increase in the power to distinguish shades. Several instances of color-blind ribbon-buyers, who were in the employ of large business houses, are recorded.

The study of the problems of color perception in hysterical conditions is difficult, owing to the great variability of words in such conditions. Then sudden variations with certain, at present, inexplicable peculiarities render the study of limited value. We find, however, in hysteria some of our best illustrations of the influence of mental conditions on color perception.

The study of children presents peculiar difficulties. Children are not always truthful, they are easily confused, and they have not infrequently misled observers. They differ greatly in color-power. Feeble-minded children show very interesting peculiarities.

March 24, 1898.

A Case of Extra-uterine Pregnancy.

H. L. WILLIAMS, M.D.

I am indebted to Mr. L. T. Salmon, of the Senior Class of the Medical Department of the University of Pennsylvania, for the history and the specimen of the present case. The ovary and ovaduct were removed by operation from a white woman of thirty-six years, with the symptoms of a ruptured extra-uterine gestation sac.

Pathological Examination.—The specimen, which has been hardened in alcohol, is about half the size of a normal ovary, and measures about seven-eighths of an inch in length, five-eighths of an inch in thickness, and three-quarters of an inch in width. From one end extends a narrow band of tissue which becomes rapidly attenuated, and to which is tied a strand of catgut, by which the specimen has been suspended in the alcohol. Upon one surface, at right angles to the long axis, a tear appears, extending nearly three-quarters of the way around the circumference of the specimen. This rupture measures one-third of an inch in width, and three-sixteenths of an inch in depth. The tissue on the bottom and sides of the tear is granular in appearance, and is the color of clotted blood. Sections were cut, and microscopic examination was first made of the tissue from the walls of the tear. Chorion villi were found in abundance, and also a small area of ovarian stroma containing Graafien follicles. When this was found the mass was cut up into eight pieces, at right angles to the long diameter, and microscopic sections made from every portion of it.

Microscopic Examination.—On examining first the small elongated attachment, to which the catgut was fastened, we find a cross section of the fallopian tube as it should appear normally at the isthmus. The branching fringes of the mucous membrane project into the lumen of the tube, the cilliated columnar epithelium is preserved, no inflammation is seen, and the tube is absolutely normal in every respect. This point is of special interest in that it shows that the retention of the impregnated ovum in the present case was not due to any obstruction to its free passage into the uterine cavity, or to an inflamed condition of the tubal mucosæ.

Examining now serial sections as they pass from the normal tube to the centre of the mass, the first deviation appears as a fibrinous coagulum of blood in the centre of the lumen. But in this section the mucous lining of the tube is still absolutely normal As we pass on, the fibrinous coagulum grows larger and larger, dilating the lumen and stretching the tube. The branching fringes become shorter and fewer in number, until they disappear altogether. The epithelium, at first columnar, becomes gradually cuboidal, then a granular layer appears, and finally a fine band that takes the stain more intensely than the other tissue, but in which no cellular outlines can be distinguished. In places the traces of epithelium are lost altogether.

No indication of inflammation is present in the tissue below the epithelium, and all the changes present give indication of being the direct *result of*, and not the *cause* of the tubal gestation. The peritoneal covering of the tube is greatly thickened, due no doubt to the peritonitis before the operation was performed. The tubal wall, at first normal, grows gradually thinner as we proceed toward the centre of the mass, until the muscular bundles finally entirely disappear, and the whole wall is transformed into a narrow fibrous band.

Webster[1] claims to have discovered portions of a tubal decidua vera in all cases of an early date examined. In the present specimen we are unable to find any trace of decidual cells in the general tubal mucosa.

Where the chorion villi came in direct contact with the tube wall, we find well developed masses of decidual cells, which in the uterus would constitute the decidua serotina.

But no decidua reflexa, such as is described by Webster[1] as existing in a specimen in the museums of the Royal College ot Surgeons in Edinburgh, can be found in our sections.

A preparation made from the quadrant situated on the side opposite the point of tubal attachment shows bands of tubal decidua serotina in some cases in contact with the ends of fixing villi, in other cases separated from them by a considerable interval, i. e., the decidual cells form the boundary toward the tubal wall of the inter-villus spaces. In every case the *villi* are covered by the syncytial layer, but in no case is the *decidua*, which forms the boundary on the side of the maternal structure, covered by this thin syncytial band.

In one place, where a fixing villus abuts against the decidual tissue, the syncytial and Langhans cells, with which the villus is covered, have been absorbed at the surface of contact.

The *origin* of the syncytial layer, which we define as the outer investing membrane of the chorion villi, made up of a homogeneous protoplasm, in which are found great numbers of nuclei with no individual cell body, is much disputed over. The question at issue is as to whether this syncytial membrane is of fetal or maternal origin.

When considered as of maternal origin, it is supposed to be derived either from the single layer of epithelial cells covering the surface of the endometrium, or tubal mucosa; or to be derived from the endothelial lining of the capillaries, which, according to Minot and Hertwig, dilate enormously to fill the inter-villan blood spaces.

When considered as of *fetal* origin, the syncytium is assumed to be a direct derivation of the fetal ectoderm.

From a careful study of the villi in the case at hand, which certainly must be at one of the earliest periods of development obtainable, we are led to draw the following conclusions:

Firstly. That the syncytial cells are not, as claimed by Ruge[2] derived from the superficial epithelial cells of the endometrium, as of course they could not be in the present case, since the villi have never been within the cavity of the uterus.

Secondly. That the syncytial cells are not derived from the superficial epithelial cells of the tubal mucosa, since the small villous buds, which cannot possibly have come in contact with the tubal mucosa, show the syncytial layer in greatest perfection.

A further argument is that in several areas, where the fixing villi are seen in contact with the tubal decidua, not only is there no trace of remaining epithelium on the surface of the decidual tissue, but the syncytial-layer which covered the end of the villus is seen to be absorbed over the area of contact, the syncytial cells, in most of the specimens, coming to an abrupt termination where they adjoin decidual tissue.

In one particular specimen, where the union between villus and decidua has evidently just taken place, the syncytial curving of the end of the villus can still be seen as in faint line, not quite yet absorbed.

Thirdly. That it would seem entirely unreasonable to accept the theory that the syncytial layer covering the villi is derived from the endothelial coat of the maternal capillaries, which expand into the intervillous space, and thus form the blood sinuses into which the villi dip ; for it would appear illogical to assume that this endothelial layer should immediately so fuse with villous surfaces that it became an inseparable part of it ever after, while on the maternal side of the cavernous blood spaces no trace of it should remain, even at the point of contact with the villi. Moreover, as the placenta proper does not form until the third month of pregnancy, and as in the specimens under examination there is no free blood present about the villi, it is evident that the placenta has not yet been formed and that the intervillous space has not yet been occupied by the expanded capillaries, so it is impossible that an endothelial covering should have been cast over the villi in the present case.

Excluding the superficial epithelium of the mucosa, and the endothelial lining of the maternal capillaries as the possible origin of the syncytium, we are forced then to accept the remaining hypothesis that the syncytial cells are of fetal origin, probably derived directly from the fetal ectoderm.

This point of apparent insignificance as to the origin of the syncytial cells, becomes of great interest and importance in considering the origin of chorio-epithelioma, or " Deciduoma malignum," by which name it was first described and is perhaps still most generally known, for it is now quite generally accepted that these neoplasms have their origin in the syncytial cells.

To sum up briefly, we come to the conclusions,

FIRST, That in the present case the changes in the tubal mucous membrane were secondary to, and the result of the detention of the impregnated ovum within the tube.

SECOND, That no tubal decidua vera was found in the case before us.

THIRD, That the syncytial layer is not derived from the superficial cells of the endometrium, or tubal mucosa.

FOURTH, That the syncytial cells are not derived from the endothelial lining of the maternal capillaries, and

LASTLY, That since the syncytial cells are not of maternal origin, they must be derived from the fetus, and probably directly from the fetal ectoderm.

(1) Ectopic pregnancy, London, 1895. (2) Gynäkologische Diagnostik, von G. Winter, 1896.

DISCUSSION.

DR. GAYLORD.—Mertens believed that the intravillous blood-spaces were formed by the expansion of the uterine veins between the villi, the endothelium of which later disappeared. Combined with this theory, which is not a new one, was the idea of some observers that the syncytium came from this endothelium. This is however clearly false, as the syncytium is formed on the villi before the opening of the uterine veins into the blood-spaces. Aschoff observed the growth of the villi directly into the uterine veins and believed that the vessels were opened up directly by the villi.

The origin of the syncytium has been a subject of ardent discussion during the past two years. Mertens, as stated, believed that the syncytium came from the maternal tissues. He observed that many villi grew directly into the uterine glands, and found syncytial-like changes in the glandular epithelium. The same phenomena have been the subject of a communication by Martin B. Smith. A somewhat similar condition has been observed in the epithelium of the duct in tubal pregnancy. Embryologists, including Minot, have held to the theory, based on observations in lower animals, that the syncytium had its origin from the foetal ectoderm. Marchand, who previously followed Mertens in this matter, has recently stated to me that his latest observations of a very young human egg have led him to accept the theory of the embryologists, and he has been able to demonstrate the direct metamorphosis of Langhans' cells into syncytium. The work of Duval is of great importance in this matter; he has been able to demonstrate that in animals the uterine epithelium disappears at the beginning of pregnancy.

DR. WILLIAMS.—The theory that the maternal capillaries of the scrotina dilate, and form the blood muscles in the intervillous space, is an old one long since advanced, but accepted at the present time both by Minot and Hertwig. Minot admits, however, that in the late stages of the placenta no endothelial lining can be detected.

The old theory that the villi grow into the glands of the endometrium has been for a long time exploded.

The theory which Dr. Gaylord mentions of the villi growing into the capillaries, opening them up, and thus allowing the blood to burst forth, is certainly of interest, but I think it can hardly be said to be very widely accepted.

Minot, although evidently widening to the belief in the fetal origin of the syncytium, does not commit himself definitely. He says: "It is still possible that the syncytium is of maternal origin, and is a grant thrown rapidly over the surface of the villi after they have been in contact with maternal tissue."

March 24, 1898.

Specimens from a Case of Pernicious Anemia.

J. A. SCOTT, M. D.

I present to-night the heart, stomach and the uterus, with its appendages, which were removed from a case of pernicious anemia about two weeks ago.

The patient was a woman aged about fifty. During her lifetime she had had repeated asthmatic attacks, which, however, had been infrequent in recent years. Last summer she had an attack of severe dilatation of the heart, since then her health has gradually declined, the urine was intermittently albuminous and high in specific gravity. On one or two occasions casts were found. The blood examined on December 20, 1897, showed red cells, 1,200,000; white cell, 6000, and hæmoglobin, 34 per cent. On January 2, 1898, the count was red cells, 1,300,000; white cells, 5000, and hæmoglobin, 25 per cent. Poikilocytosis was marked, and macrocytes and microcytes were observed.

On January 31 and February 10 the hæmoglobin was reduced to 20 per cent. There had been some nausea and other symptoms referable to the stomach. On December 22, 1897, the stomach contents were examined. There was no free hydrochloric acid, a trace of lactic acid was present. Microscopical examination showed a few red and white blood corpuscles. A few weeks before death a small mass was discovered in the left, lower zone, and upon abdominal and vaginal examination a large hard mass detected in Douglas' cul de sac. The anæmia and prostration were so extreme that no operation therefor was suggested.

The vascular phenomena were most marked, and murmurs were heard over all the cardiac areas. With each systole the veins of the neck would pulsate up to the angle of the jaw. At intervals the liver pulsated. Her distaste for food became extreme, and on February 22 the patient died of asthenia.

The autopsy was made about twelve hours after death. The body, especially the hands and feet, was extremely yellowish-white in color. There was slight œdema over both tibiae. There was little or no superficial fat upon incision through the chest wall and abdomen; the muscles were deep red in color. There was possibly a pint of dark almost bile stained yellowish fluid in the abdominal cavity. There was about the same amount of fluid in the pleural cavity. Both lungs were marble white in color, very emphysematous, especially about the anterior margins. There was considerable œdema, with slight hypostatic congestion in both bases. There was about one ounce of clear and dark yellowish fluid in the pericardial sac. The right auricle extended about one inch and one-half to the right of the midsternal line. It contained a small amount of thin watery blood. The right ventricle was distended, relaxed and contained a few light colored "currant jelly" clots. The thickness of the right ventricle was three-sixteenths of an inch. The tricuspid valve admitted the tips of the thumb and three fingers. The left ventricle was contracted and contained no blood; its wall measured ten-sixteenths of an inch. The mitral and aortic valves were competent. There was one small calcareous nodule on the posterior aortic leaflet. The cardiac muscle was extremely fatty, and showed marked mottling, especially of the interventricular septum. There was no general atheroma of the blood vessels.

The stomach, which lay diagonally within the abdomen, resembled, in its appearance, the large intestine. It seemed to be hitched up at the pyloric extremity by an attachment to the liver, which was low in its position. It measured about an inch and one-quarter from the cardiac to the pyloric orifice. It weighed five ounces. Its walls were much thickened. At the cardiac extremity, the measurement, including the peritoneal coat, was five-sixteenths of an inch. The pyloric extremity four-sixteenths of an inch. There was considerable pigmentation, of a dark brown color, on the tops of the rugæ in the neighborhood of the pylorus. The interesting features of this stomach are its unusual narrowness, its small capacity, and the thickness of its coats. I think it can be termed fibroid or cirrhotic. The intestines were not examined.

The kidneys were about normal in size. The capsules were adherent, in stripping some of the kidney substance was removed, and the surface was distinctly granular in appearance. The cortices were, to the naked eye, not materially reduced in size. They presented a pallid, pale flesh color. The liver was interesting in its appearance. Upon opening the abdomen, the right lobe extended to the median line, and down as far as the umbilicus. It was rather light yellowish in color, its consistency seemed increased, and there were minute dark areas over the entire surface and upon sections of it. It was both fibroid and fatty, and in appearance resembled the so called "nutmeg liver." The gall bladder was tightly distended with bile, the ducts were patulous; no stones were found. The uterus was somewhat atrophied. At either horn there was a small extramural fibroma. Growing from the right fibroma was a third tumor measuring three by three inches, which hung by a small pedicle down behind the uterus well into the pelvis; it was pearly white in appearance, and evidently a hard fibroma. There was a small cyst in the left ovary. The ovaducts seemed normal.

The etiology and morbid anatomy of pernicious anæmia have been closely studied. The skin is usually extremely pale or of a lemon yellow color, so much so that the patient looks jaundiced. The muscles are commonly intensely red. There is usually little or no wasting, though sometimes there is a moderate amount of atrophy or emaciation. The subcutaneous fat may be small in amount and light yellow in color. Subcutaneous hemorrhages are common, the serous and mucous membranes are also affected. The amount of blood is probably diminished; post-mortem there may be little or no blood found in the heart. Microscopically, the blood in these cases shows a marked reduction of the red corpuscles, frequently below 1,000,000 per c.mm. Quincke reports one case where the count was 143,000. Stengel remarks that the blood count is frequently reduced to 1,000,000 corpuscles, and then the subsequent reduction takes place very slowly. I have seen on three occasions blood counts of 650,000, 600,000 and 835,000 per c.mm. The hæmoglobin is likewise reduced, but may be relatively increased in proportion to the number of cells, each corpuscle containing about one-seventh to one-tenth more than does the normal blood corpuscle. Megalocytes form about one-eighth of all corpuscles, ranging in size from 8½ to 16 *v*. Poikilocytosis is common, and nucleated red blood corpuscles are quite a constant feature. The leucocytes play but an unimportant part in this disease, though toward the termination of the process they may be markedly increased.

There is nothing unusual to be observed in the examination of the lungs. The heart is always the seat of a marked fatty degeneration affecting the muscle wall in such a way as to produce the appearance known as "wren's breast," or "tabby mottling." Frequently one or other of the cavities may be dilated.

The liver is usually very fatty, it mav sometimes be large and at other times the size is not altered. In 1876, Quincke called attention to the fact that in pernicious anæmia there was a deposit of iron pigment to be found in the liver. Later William Hunter demonstrated the condition conclusively. A deposit of iron takes place in the median and outer zones of the lobule. In forty-seven consecutive autopsies at the Johns Hopkins Hospital I did not find this pigment so deposited except in pernicious anæmia.

The kidneys are usually fatty. They sometimes contain pigment. There is no change commonly observed in the spleen, though some observers have considered that it is the seat of the morbid cell destruction.

The stomach is sometimes the seat of marked glandular degeneration and atrophy of the muscular coat; in the latter case the walls are thin. Another condition that has been described is fibrosis, an over-growth of the connective tissue, a submucous hyperplasia. There is no special change found in the pancreas.

In recent years some interesting observations have been made upon the spinal cord. Burr has confirmed the descriptions of degeneration of the posterior and lateral columns, which is found especially in the cervical regions. The bone marrow presents some very interesting changes. There is usually a change from the yellow fat color to rather a reddish tinge, the embryonal appearance. These changes are best seen in the long bones. Microscopically, a disappearance of fat globules is noted, with an enormous increase in the nucleated red corpuscles. In addition to these there are many non-nucleated forms of large size. It is to be observed, however, that similar changes are to be met with in the bone marrow in diseases other than pernicious anæmia, and that they are probably the results rather than the cause of the anæmia.

Pernicious anæmia was first looked upon as of hæmogenetic origin. Since, however, Quincke's observation in 1876, and the further investigations of Hunter, our views have changed as to the disease originating in that manner. Hunter's views as to the hæmolytic origin of pernicious anæmia are now generally accepted by the profession.

The seat of this hæmolysis is still an open question. Some observers consider the liver, others the spleen, the seat of the blood destruction. The agreement among most recent investigators on this subject is that the disease is a toxic hæmolysis produced by intestinal auto-intoxication.

March 10, 1898.

A Case of Aneurysm of the Aorta with Rupture Into the Right Pleural Cavity.

JOSEPH McFARLAND, M. D.

There is, unfortunately, no clinical history of the length and course of the disease. The only known facts of the case are that the patient felt unwell, and applied for help at one of the dispensaries of the city, and fell dead there while talking to one of the officials.

The patient was a woman apparently about sixty to sixty-five years of age. She was considerably emaciated, the surface of the body was very anemic. When I saw her first at the autopsy, rigor mortis had disappeared; the cadaver was in fact in process of decomposition, and the belly was already somewhat green. The body was opened by the usual incision. Upon removing the sternum and costal cartilages, serum was observed to escape as if there were an effusion in the right pleural cavity. As the sternum and costal cartilages were cleared away, however, it was found that the pleural cavity of the right side was filled with blood, apparently of recent escape from the vessels and freshly coagulated. The pleural cavity was absolutely full of blood so that the lung was compressed against the superior posterior part of the chest cavity. There were a few old pleuric adhesions at the central part of the lung and here and there on the posterior surfaces. These adhesions were of some interest as they were in condition of hemorrhagic infiltration and were dark red and gelatinous in appearance. The lung was so compressed as to crepitate but little. When the blood clots were removed it expanded again.

The blood was removed from the pleural cavity and its source sought for. Nothing was apparent except a solid body about the size of a horse-chestnut, situated at the anterior, inferior, internal part of the lung, not definitely attached to it, but also adherent to the pericardium. The body resembled a neoplasm somewhat, and had at its free surface what seemed to be a laceration. The nature and attachments of this body were not then apparent.

Upon attempting to remove the heart, it was found that the pericardium was everywhere adherent to that viscus, so that its removal by the ordinary methods was impossible. Fearing that some interesting aneurysmal lesion might be destroyed by careless dissection, I removed the entire thoracic viscera in toto, and made a subsequent dissection of them.

The heart is not hypertrophied. It was soft and flabby. The right venticle was entirely empty, its orifices and valves normal. The left ventricular cavity was likewise empty. Its valves showed some beginning sclerosis. The auricles were normal and empty.

The aorta was very sclerotic, and at the ascending part of the arch underwent an aneurysmal dilatation the size of a man's fist, which projected downward, forward and outward. At the apex of the dilatation there was a rupture through which the finger could easily be introduced. It was from this opening that the woman bled to death into her own pleural cavity. The discovery of this rupture made clear also the nature of the neoplastic mass first encountered when the lung was examined. At the apex of the aneurysm there seems to have been a kind of sacular diverticulum in which fibrin formation had progressed for a time. The form ot the rupture had everted this mass through the perforation.

The right lung was compressed. The left lung was everywhere thoroughly crepitant. Both lungs contained a few fibrous nodules. It is scarcely necessary to mention the details of the study of the other organs as, except for profound anemia and a slight degree ot fibrosis, they were without apparent pathological change.

The case represents one of the most frequent forms of aneurysm of the aorta and also one of its frequent terminations.

Crisp collected 234 cases of aneurysm of the aorta, of which 175 (74.8 per cent) affected the thoracic aorta; 59 (25.2 per cent) affected the abdominal aorta and its branches.

Of 167 aneurysms of the thoracic aorta studied, 98 (58.8 per cent) were of the ascending arch; 48 (28.7 per cent) were of the arch itself; 21 (12.6 per cent) were of the descending part of the arch.

Discussion.

Dr. J. A. Scott emphasized the fact that in many instances of aortic aneurysm recently reported, and in others lately exhibited here, hypertrophy of the heart has been but infrequently observed.

Dr. Riesman called attention to frequency of multiple aneurysms, either in the same trunk or in the arterial tree.

March 24, 1898.

A Dermoid Cyst Obstructing Labor and Requiring Cæsarian Section.

H. L. WILLIAMS, M. D.

This case occurred at the University Hospital, in the service of Dr. B. C. Hirst, to whom I am indebted for the privilege of showing the specimen this evening. The history is as follows:

Mrs. H., aged twenty-three, was married three years ago, and became pregnant for the first time in June, 1897. Her menstrual history began at the age of fourteen. The periods were regular, lasting from five to seven days, and were painful only during the first day. Since marriage the periods have been regular, but have lasted about ten days. She has also suffered much pain during the first days, and says that she has lost large quantities of blood. Her last menstruation occurred in the middle of May, 1897.

The history of the case, before this time, bearing upon the present trouble, was as follows: Two years ago a physician in this city was consulted on account of the increased amount of pain at the menstrual period and the unusual loss of blood. A thorough examination was made and a large tumor discovered, which was supposed to be a fibroma.

At this time she also suffered from bearing-down sensations. backache, and frontal headache. Until the examination was made the presence of a tumor was unsuspected.

The early months of pregnancy were without incident, except that on two occasions she had slight discharges of blood. As gestation advanced, it became evident that normal labor would be impossible, on account of the tumor obstructing the outlet of the pelvis, and about six weeks before full term the patient was sent to the maternity ward of the University Hospital.

The woman was then seen by Dr. Hirst, and after a careful examination, in which the pelvis was found to be completely obstructed, the operation of Cæsarian section was decided upon. This operation was performed on February 8, 1898, two weeks before expected labor. The usual section was made, and a living child delivered without difficulty. It was then found that the pelvic obstruction was caused, not by a fibrous tumor, but by a large dermoid cyst of the left ovary, which was removed and is here presented. When first taken out the cyst was larger than the foetal head, and its shape closely resembled that of a turtle's egg. Its polar diameter measured eight inches, and its equatorial circumference about twelve inches. On section a soft cheesy matter exuded, which upon examination was found to constitute almost the entire bulk of the specimen. Among the sebaceous debris was found a flattened plate of bone, and a considerable quantity of hair. But no teeth were apparent, or any other structures that could be identified. *March 10, 1898.*

Rupture of Kidney and Spleen.

W. E. HUGHES, M. D.

These organs were removed from the body of a man crushed between cars. Immediately after the injury he became profoundly shocked, and soon began to void urine looking almost like pure blood. This bloody urine continued for eighteen hours, after which it became clear and remained free from blood. He developed peritonitis and died three days after the injury. The kidney is torn completely through into the pelvis, but in neither pelvis nor uretes is there any blood. The renal vein is completely occluded by a firm thrombus, the artery is free. In the connective tissue about the kidneys there is a large extravasation of blood. The peritoneum is not torn. The peritoneal cavity contains some deeply blood-stained serum. The coils of intestines are everywhere bound together by recent lymph. In the spleen is an extensive tear perfectly filled by recent clot.

This case follows several others, which I have seen, in having a much freer hemorrhage from the torn kidney than from the spleen The peritonitis was probably due to the rupture in the pelvis of the kidney though there was no rupture of the peritoneum.

Heart and Lungs from a Bicycle Rider.

W. E. HUGHES, M. D.

A boy of nineteen was thrown from his bicycle while coasting, sustaining a fracture of the base of the skull. He died of cerebral meningitis. Previous to his accident his health had been uniformly good. He had used his bicycle constantly but apparently, at least in his own estimation, to no injurious extent.

The walls of the left ventricle were thickened and the cavity enlarged. The muscle was normal in appearance. At the base of the aorta and on the anterior leaflet of the mitral valve were several spots of soft atheroma.

The bases of both lungs on the latero-posterior aspect showed a most unusual sub-pleural emphysema. This emphysema was arranged in lines in an arborescent manner with the branching extremities pointing away from the root of the lung. These lines of emphysema apparently ran along the vessels.

With the exceptions of a basilar meningitis the other organs were healthy.

In the absence of any visceral disease, especially of kidneys, I am compelled to regard the cardiac condition as due to bicycling. This, too, may possibly have been the cause of the emphysema.

Pericarditis in Bright's Disease.

W. E. HUGHES, M. D.

I present this heart in connection with my remarks upon the Pericardium made at the last meeting. It shows well the fibrinous pericarditis, which is the commoner form accompanying Bright's disease. It was obtained from a negro woman, aged fifty years, who died of uraemia after a history of chronic Bright's disease, the prominent symptoms of the uraemia being prolonged epistaxis and vomiting. There were no other reasons for the pericarditis nor did it give rise to any symptoms, being recognized only by physical examination.

Discussion.

Dr. J. A. Scott.—Absence of blood from the pelvis of the kidney is very rare following a rupture of the organ with hematuria.

Dr. Riesman.—As to the atheroma present in the aorta of the young man, while I consider the over-exertion to have been the most likely cause, it is obvious that infectious processes have not been excluded. In regard to the non-occurrence of extensive hemorrhage after rupture of the spleen, might it not be explained by the elastic retraction of the capsule of the organ? Had the kidney such a fibro-elastic capsule the hemorrhage might be controlled.

March 24, 1898.

Aneurysm of the Arch of the Aorta. Hour-Glass Stomach. Marked Fibroid Myocarditis, with Extensive Valvular Contraction. Sarcoma Presenting Many Mastcells.

A. E. TAYLOR, M. D.

The aorta presents the following conditions: A fusiform aneurysm springs from the ascending arch directly above the valves; from this springs, at the superior aspect of the transverse arch, a sacular aneurysm, which extended upwards into the neck, was partly filled with a firm laminated clot, and presents a rupture at the uppermost point through which the patient had bled to death, the cutaneous opening being at the right sternal sterno-cleido-mastoid articulation; beyond the transverse arch the aorta presents a second fusiform dilatation, below which the vessel is most markedly atheromatous. The heart is not obviously hypertrophied; the valves of the left heart are contracted and sclerosed; the peripheral arteries were also extremely calcareous. The subject had had syphilis. The liver was cirrhotic and fatty, the pancreas was very indurated, the kidneys were contracted, the prostate was hypertrophied, and the bladder was thick and contracted. The age of the patient was not over fifty years.

The hour-glass stomach was removed from the same subject as the aneurysm previously described. There is a firm fibrous stricture four inches above the pylorus, the opening is about the same as the lumen of the normal pylorus. The pyloric orifice is constricted to one-half its normal lumen. There are no macroscopic signs of malignant disease. The gastric mucosa is atrophic, the stomach wall and the muscularis do not seem hypertrophied, the viscus is not dilated.

This heart was removed from the body of a man with interstitial nephritis. It illustrates the pure hypertrophy of the left ventricle sometimes seen in connection with Bright's disease. The aortic valves are markedly contracted and moderately thickened; the mitral valve is soft and seems competent.

The sections of sarcoma were cut from a growth which involved the sacral and pelvic regions. The neoplasm is of the spindle-celled variety. Throughout the tumor are myriads of mastcells, often clumped along the strands of fibrous tissue, and about the blood spaces. In the stained specimens the collections may be seen with the naked eye. No stains bring out these granulated cells as well as thionine and tuloidin blue; the counterstain in these thionine preparations is acid fuchsin.

March 24, 1898.

Hepatic Embolus in the Lung. Illustrations of Cardiac Lesions.

H. GAYLORD, M. D.

The subject from whom the embolism was secured was killed by a crush upon the chest and abdomen, dying twenty minutes after the accident. The autopsy was performed two hours after death. The embolus of hepatic tissue here exhibited was discovered in the lung; there was also hepatic tissue in the heart. In the sections the hepatic cells are seen to be greatly compressed. The elastic fibres in the walls of the vessels about the area are seen to be ruptured. This I consider the result of the traumatism rather than of the infarction. Cellular emboli in the lung are uncommon. The two other varieties are the placental and myelogenous depositions; they are likewise commonly the result of traumatism.

I present for inspection a set of Schmoerl's stereopticon views illustrating the various lesions of the heart. These most accurate and beautiful reproductions are soon to be the material of a special publication by the author. *March 24, 1898.*

CARD SPECIMENS.

Dr. Fisher presented the specimens of an aneurysm which had caused death by rupture into the left pleural cavity. The aneurysm was of the dissecting variety, and sprang from the transverse arch of the aorta, which was slightly dilated and presented numerous areas of atheroma. The heart was hypertrophied, the valves were not sclerotic; the kidneys were contracted

Dr. Chestnut presented an eye of a calf, with a large heavy tuft of black hair springing from the cornea. The entire surface of the cornea was not involved. The other eye of the animal was similarly affected. *March 10, 1898.*

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TABLE OF CONTENTS.

JUDSON DALAND, Aneurysm of the Arch of the Aorta Causing Death by Rupturing into the Left Bronchus,—ARTHUR V. MEIGS, Demonstration in Sections of a Human Heart, of which the Arteries Were Injected, of the Presence of Capillaries within the Muscular Fibres; and of the Absence of Veins of the Smallest Size from the Heart, —H. L. WILLIAMS, Uterus Subseptus.

Aneurysm of the Arch of the Aorta Causing Death by Rupturing into the Left Bronchus.

JUDSON DALAND, M. D.

The patient from whom this specimen was removed was a man aged thirty-five years, a driver by occupation. There is nothing of importance in the family history excepting the deaths of two aunts and two uncles from pulmonary tuberculosis. He suffered the ordinary diseases of childhood and occasionally had attacks of malaria. He acknowledged five attacks of gonorrhœa, and was infected by syphilis at the age of twenty-three. He drank beer to excess and frequently became intoxicated. He began the use of tobacco at the age of six years and chewed and smoked to excess.

When first examined, on December 8, he complained of pain in the chest, numbness of the legs, a metallic cough of one year's duration, and the expectoration of about half an ounce of white, frothy, viscid sputum daily.

Physical examination showed enlargement of the heart and the displacement of the apex downward to the sixth interspace. A second area of pulsation, having a diameter of two inches, was discovered in the third interspace in the left mid-clavicular line, and auscultation at this point revealed a systolic murmur. Further examination showed evidences of considerable pressure upon the left bronchus, with a diminution of the supply of air in the left lung.

There was no tracheal tugging, no thickening of the walls of the radial arteries; the left pupil, on two examinations, was larger than the right.

On January 28 he died suddenly from hemorrhage into the left bronchus.

The aneurysm is sacculated, about the size of an orange, and the rupture into the left bronchus is seen as a slit having a length of half an inch. The walls of the aorta show a moderate grade of atheroma. The heart shows no change in size nor any alteration in the cavities, orifices or valves.

The case is interesting because of the absence of tracheal tugging, which would have been expected from the situation and direction of the aneurysm; the comparative early age of the patient; the existence of chronic alcoholism and the occurrence of syphilitic infection ten years previously. *March 24, 1898.*

Demonstration in Sections of a Human Heart, of which the Arteries Were Injected, of the Presence of Capillaries within the Muscular Fibres; and of the Absence of Veins of the Smallest Size from the Heart.

ARTHUR V. MEIGS, M. D.

In two respects the blood-vessels of the heart differ from those of the other organs. This I pointed out in a paper read before the College of Physicians of Philadelphia in the year 1891.* When my original communication upon the subject was read, it was suggested by someone in the discussion which followed that injection preparations ought to be made to furnish additional proof of the correctness of my statements. My reply was that the sections of tissue which were then shown, demonstrated so thoroughly the anatomical peculiarities that they should not be doubted, but that it would be useful to have injection preparations for the satisfaction of any one who might not be convinced. Such preparations I am glad to be able to show to the members of this society to-night. The anatomical peculiarities are seen exactly as they were previously described, and, so far as I have been able to ascertain, the preparations show nothing more. The result of the examination of the injected tissue is therefore merely the addition of another link to the chain of proof. The marked contrast between the blue color of the vessels which were filled and the red of the carmine with which the rest of the tissue was stained, makes the blood vessels more easy to recognize.

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*Transactions of the College of Physicians of Philadelphia, April 1, 1891, and the American Journal of the Medical Sciences, June, 1891.

The two peculiarities of the blood vessels are: First, that there are no minute veins with three coats in the deeper portions of the muscular substance of the heart, but in their places are vessels having the structure of capillaries; and, Second, the capillaries of the heart do not simply lie adjacent to and surround the muscular fibres, but penetrate them so that the capillaries can be seen even in the centres of the fibres. These two peculiarities are shown by the sections of injected heart exhibited, which are from a man thirty years old who died of lead encephalopathy. The heart was of normal size and appearance, and at the time the autopsy was made the two coronary arteries were injected with a solution of Berlin blue (Grübler) of the strength of three grammes to six hundred cubic centimeters of water. The heart was at once cut in various directions and pieces of suitable size were placed in seventy per cent. alcohol for preservation. The tissue was stained in bulk in borax-carmine and embedded in paraffine.

The statement that minute veins are absent from the deeper portions of the muscular substance of the heart requires explanation. It is well known that upon the surface and in the connective tissue interstices of the heart, arteries are found with their venæ comites just as they are in other organs and tissues. In marked contrast with this, however, is the condition in the muscular substance of the heart. When an arteriole has an efferent vessel with it, it is single-coated and composed of endothelium exactly like the smallest capillaries, except for its greater size. As the arterioles in any tissue break up into smaller twigs they are less frequently found in company with efferent vessels, the arrangement that the afferent and efferent vessels lie together being one of convenience when the blood is traveling to and from parts to which it is to be supplied. Consequently the smallest arterioles in all tissues are often unaccompanied by veins. The large efferent capillaries of the heart that have been mentioned are therefore generally without accompanying arterioles. These capillaries are so numerous and of such size that it seems likely they perform the functions of reservoirs. Their presence indicates a provision for blood supply even more bountiful than that in the other organs.

With regard to the relation of the capillaries to the muscular fibres, the sections exhibited show distinctly that the fibres are often penetrated by capillaries which can be seen in all possible situations within the fibres. Sometimes the capillaries slightly indent the sides of the fibres, again they lie within them but close to their peripheries, and in some instances capillaries are seen in the very centres of the fibres. It is, of course, impossible that the capillaries are within the muscular cells at an early embryological stage. The condition must therefore be one of later development, and as yet it is not known when it begins to exist. It would be interesting and useful to examine young human hearts to ascertain at what age capillaries are first found within the muscular fibres. This aspect of the subject was alluded to by the late Dr. John A. Ryder in discussing my original communication. Amongst other things he said:

"Many of the structures that we see in the adult organ are really developed during what embryologists speak of as the post-embryonic period. When we reflect upon the way in which muscle universally develops, no matter whether it be in the salamander, the fish, reptile, bird, or mammal, we can see how, in the later stages of the development of the heart, such a thing as enveloping or inclusion of capillaries might occur. It is well known to everyone who has made a cross section of a developing muscle, that the fibrillæ appear as delicate threads imbedded in the protoplasm forming the body of the muscle cell. That is the type found in the highest form of muscular development. There are forms of muscular development in which the fibrillæ are developed in a continuous roll, and, as a result, the muscular fibre resembles a flat plate made up of parallel filaments. However, as we pass up in the animal scale, and particularly in mammals and birds, the muscular fibres become irregular in shape and excessively numerous and closely packed together, with very little protoplasm between. When we remember that the multiplication of these fibrillæ seems to result from splitting up or multiplication of the fibres already present, we can understand that, as the muscular fibres grow in thickness, any capillaries that might lie beside them might readily be engulfed and covered in by the muliplying fibres."

In a paper on the Histology and Histogenesis of the Heart-Muscle Cell, by John Bruce McCallum (*Anatomischer Anzeiger, Centralblatt für die gesamte wissenschaftliche Anatomie*, XIII. Band, No. 23, 1897) it is clearly shown that during early embryological stages the muscle-cells are composed of undifferentiated sarcoplasm, even after the heart has begun to beat, in the case of the chick. The appearance of the fibrils with their characteristic cross striæ comes at a later period, and shows itself first in the more superficial layers of the heart. During the early stages it is therefore evident that the capillaries are not within the muscle-cells. McCallum quotes Schiefferdecker as saying that, "after the tenth year yellowish-brown pigment is present at the poles of the nucleus in human heart muscle. As age advances the pigment increases in quantity, and the granules become larger in size and of a deeper color."

In the future it must be remembered that all of the nuclei in the muscular fibres are not muscle nuclei, but that the capillaries in penetrating the fibres necessarily carry their own nuclei with them into the fibres. Therefore, some of the nuclei which are seen in the fibres are the endothelial nuclei of the capillaries. This peculiarity is distinctly shown by the sections which are exhibited.

The penetration of the muscular fibres by the capillaries must have important pathological bearings, but at present little can be said about it. It is extraordinary how little is as yet known in regard to disease of the capillaries. It is known that they constitute the link connecting the arterial and venous systems, and we have a more or less complete knowledge of their ramifications and structure, but their pathology offers an almost untrodden field for the future investigator, and one of vast importance.

April 14 1898.

Uterus Subseptus.

H. L. WILLIAMS, M. D.

The uterus subseptus here presented was removed at the University Hospital in December, 1897, by Dr. B. C. Hirst, to whom I am indebted for the privilege of exhibiting it. The patient from whom the specimen was taken was a young woman, twenty years of age, with acute septic salpingitis and oöphoritis of gonorrheal origin. Previous to the acute attack for which operation was performed the woman had given birth to one child, and had had a miscarriage at three months in the summer of 1897. The clinical symptoms and history were those of the ordinary case of gonorrheal salpingitis and oophoritis, the interest in the case centring entirely in the anomalous condition of the uterus which was discovered upon examination after removal. At the time of operation the fundus was found to be so broad and the walls of such unusual thickness, that, in the light of the clinical history, it was believed that the uterine wall had become septic and was probably the seat of mutiple abscesses, hence the hysterectomy.

Pathological Examination.—Measured externally, the width of the fundus was 8½ cm., and its length, to a short distance below the internal os, where amputation was performed, was 7½ cm. The antero-posterior diameter measured 6 cm. Both tubes contained pus and were considerably enlarged, with patulous osteii. The ovaries were enlarged and covered with inflammatory adhesions. The right tube measured 9 cm. in length and 1¼ cm. in thickness at its greatest diameter. The right ovary measured 7 cm. x 2½ x 2 cm. in its various diameters. Upon the left side the tube measured 10 cm. in length and 2 cm. in its greatest diameter. The left ovary measured 5 x 2 x 1¾ cm. and was the seat of numerous follicular cysts. The uterus was then opened to the extremities of the cornua by two lateral incisions along the lines of the broad ligament, and the anomalous conditions present made apparent.

Previous to the opening of the abdomen, the uterus had been curetted. On examining the specimen the cavity is seen to be triangular in shape, with cornua greatly lengthened and enlarged, diverging at an angle of 75° and separated by a wedge-shape septum, which extends to a point situated more than half way from the fundus to the internal os.

From the lower point of the septum to the point of tubal attachment the cornua measured 3 cm. in length. The width of the right cornual cavity measures 1¼ cm. From this horn the mucous membrane has been for the most part removed by the curette, though the lining membrane of the cornua upon the opposite side remains undisturbed.

Upon the left side the cornual cavity is of the same length, but the lumen measures only ¾ cm. in diameter. From the lower extremity of the septum to the outer surface of the fundus, the tissue measures 3¼ cm. in depth. The lateral walls of the uterus measure 2 cm. in thickness, while the uterine cavity just above the internal os is only 1¾ cm. in width.

The etiology of malformations of the uterus furnishes a most interesting study. Anomalies of the uterus are due in every instance to arrested development during the early months of fetal life.

Until the classical papers by Küssmaul in 1859, Fürst in 1867, Jo Dohrn in 1869, the etiology of malformations of the uterus was not understood.

Küssmaul demonstrated that the term "malformation" as generally used, is a misnomer;—that the parts present in the anomaly are not misformed, but that the development of the organs has been arrested. The *time* at which arrest of development takes place determines the character of the anomaly.

This is determined by a study of the embryogenesis of the female generative apparatus. To summarize briefly:

The genital cord contains four ducts, viz., the two Wollfian ducts and two Müllerian ducts, all parallel, the latter being placed close to one another and slightly posterior.

In the female sex the Wollfian ducts atrophy while the tubes, uterus and vagina are all developed from the Müllerian ducts.

According to Fürst, fusion begins at the end of the eighth week of embryonal life at a point two-thirds of the distance from the cephalic end of the ducts to the uro-genital sinus.

The cephalic extremities remain permanently separated and form eventually the Fallopian tubes. At first separated by an acute angle, they grow rapidly and become horizontal to the axis of the duct at about the sixth month of fetal life. According to Fürst, the septum between the Müllerian ducts remains unbroken until the eighth week.

Between the eighth and twelfth weeks, the septum between the fused portions of the ducts disappears, forming the cavity of the uterus in the upper part and the vagina in the lower.

During the fourth and fifth months the angle between the unfused, upper extremities of the ducts becomes greater and greater, until they finally occupy a position at right angles to the axis of the ducts, and develop into the Fallopian tubes, as above stated.

During the last four months of uterine life the flattened end of the uterus becomes arched by the development of the fundus.

It is therefore readily seen that arrest of development, either of the entire Müllerian ducts, or any part of them, during the formative period, may produce an anomalous condition.

Complete absence of the uterus may occur, though rarely. This can only be demonstrated post mortem, as in nearly all instances a small band of muscular fibres and connective tissue is found upon the posterior wall of the bladder.

In the uterus bipartitus rudimentary horns, which may be either solid or hollow, are present, while the ducts below the points of fusion become a cicatricial band.

Should the septum persist throughout the length of the Müllerian ducts and the two develop separately, the condition known as " uterus bicornis duplex " exists.

If the lower portion only, of the ducts fuse, forming a single vagina, while the upper uterine portions remain separate, we have the uterus didelphys.

Should one of Müllerian ducts be arrested and the other develop, the condition of uterus unicornis is present.

The condition in which the septum between the portion of the ducts forming the cavity of the uterus persists is known as the "uterus septus," while if only the upper portion of the uterine septum persists, we have the so called "uterus subseptus" as seen in the specimen presented this evening.

April 14, 1898.

Proceedings
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TABLE OF CONTENTS.

RIESMAN, Meckel's Diverticulum and the Omphalomesenteric Duct.—**HARRIS**, Two New Methods of Staining the Axis-Cylinders of Nerves in the Fresh State. Some Microchemic Reaction of Toluidin Blue.—**SAILER**, Nucleolar Degeneration of the Ganglion Cells.—**HARE AND HARRIS**, Miliary Carcinomatosis of the Peritoneum, with Microscopic Slides Showing so-called Micro-organisms of Cancer.—**HARE**, Specimens of Cardiac Lesions.—**HARE**, Spleen and Liver from a Case of Leukaemia. **PACKARD**, A Case of Gall-Stone in the Common Duct, with Intermittent Fever.—**GITTINGS**, A Case of Acute Appendicitis with Secondary Pylephlebitis and Multiple Abscesses of the Liver.—**PEARCE**, A Case of Intracanalicular Fibroma of Both Mammary Glands.—**STEELE**, Rupture of the Aorta, with Flap-Like Dissection.—**TAYLOR** Obliteration of the Orifice of the Right Coronary Artery.—**TAYLOR**, Multiple Abscesses of Lung following Pneumonia and Empyema: Ulcerative Endocarditis.—**MORGAN**, Cancer of the Caecum.—**PEPPER**, A Case of Cancer of the Rectum and Liver.—**PEPPER**, Obstruction of the Cystic Duct.

Meckel's Diverticulum and the Omphalomesenteric Duct.

FROM THE PATHOLOGICAL LABORATORY OF THE UNIVERSITY OF PENNSYLVANIA.

DAVID RIESMAN, M.D.

In an autopsy that I recently had occasion to perform at the University Hospital on a man, aged 31 years, who had died of exhaustion following chronic colitis, I found, on opening the abdominal cavity, a long, fibrous cord passing from the peritoneal surface of the umbilicus towards the right iliac fossa and joining a coil of small intestine On dissection it was seen that the place of insertion of the cord was on the side of the lower ileum, near the mesentery, at a point three centimeters above the ileocecal valve. The cord was eleven centimeters long and about three centimeters in width, cylindric in form, and apparently solid throughout. At its points of fixation is was a little wider than along its course.

The mucous membrane of the ileum corresponding to the point of insertion was perfectly smooth and showed no opening into the cord. Beyond its two attachments—to the umbilicus and ileum—the band had no other point of fixation. During life it must have acted as a sort of suspensory ligament of the intestine, and must have been subjected to more or less traction when the patient was in the upright posture.

There was some adhesive inflammation at the ileocolic junction probably connected with the extensive ulceration present in the cecum and colon, the intestines being the seat of a very marked colitis polyposa. This latter feature of the case will be dwelt upon in a separate paper by Dr. Stengel.

The cord itself seemed to have produced no bad effects of any kind.

It is interesting to note that the man had other congenital anomalies, the most striking of all being a marked hypoplasia of the lungs, both organs together weighing only 520 grammes, or seventeen ounces, as against forty-two or forty-three ounces, the normal weight. As there was no apparent pulmonary disease, and as the man was only 31 years old, the smallness of the lungs could have had none other than a congenital cause. There was, furthermore, a supernumerary spleen, and the remains of the hypogastric artery on the right side were remarkably conspicuous, the cord representing the obliterated artery being unusually thick and readily traceable up to the umbilicus.

As to the nature of the fibrous cord first described, there can be no doubt that it represents the remains of an embryonal structure, the omphalomesenteric or vitelline duct, or the vessels that in embryonal life accompany it. As will be shown below, there are reasons for considering it the remains of the vessels.

The vitelline duct is a tubular canal which at a very early period in embryonal life connects the primitive gut with the vitelline or yolk-sac. In the beginning, it will be remembered, the embryo lies with its endodermic surface spread out, like a leaf, on the yolk-sac. By a curving inward of the cephalic and the caudal extremities it becomes differentiated from the latter, and, the sides also folding in, is gradually lifted above the yolk-sac. The closure of the lateral folds forms the primitive gut, which, however, remains connected with the yolk-sac by means of the vitelline duct, along which run the vitelline vessels, the two, the duct and the vessels, forming constituent parts of the umbilical stalk. Examined at the end of the fourth week of embryonal life, the communication between the gut and the now-atrophied yolk-sac is only a narrow canal. At the beginning of the second month the intestinal navel closes entirely and the vitelline or omphalomesenteric duct becomes obliterated. For a short time a loop of gut is still partly contained in the umbilical cord, but in the middle of the second month it withdraws from the latter, and the intestines then lie free in the abdominal cavity.

If the processes of constriction and retraction are interfered with, various malformations may result, consisting in a persistence of parts or the whole of the omphalomesenteric duct. We may classify the resulting anomalies as follows:

(1) Persistence of the proximal part of the duct,—Meckel's diverticulum.

(2) Persistence of the entire duct as a patulous canal.

(3) Persistence of the distal part of the duct, that connected with the navel.

(4) Persistence of a part of the duct near its middle.

(5) Persistence of the omphalomesenteric vessels.

Much evidence exists that these omphalomesenteric remains are capable of producing more or less serious disturbances; indeed, many fatalities are attributable to them. As to the frequency with which the anomalies are found, we have the statistics of Deschin,[1] who noted vestiges of the vitelline duct in twenty-five out of 1367 bodies, or in 1.8 per cent.

In the lower animals remains of the duct are quite common. Allen[2] found them in every new-born cat and dog up to the age of a fortnight or more, and in a young lion of seven weeks. In a human fetus three inches in length they were of an extreme degree of tenuity. Schroeder[3] also found such vestiges in new-born cats, dogs, and rabbits. Persistence of omphalomesenteric remains in the form of a diverticulum is, as Cazin[4] has shown, the rule in the lower orders of birds, such as the water-fowl, snipe, and swan; in some species of the ostrich family the diverticulum is replaced by a sac containing altered vitelline substance.

Fig. 3.

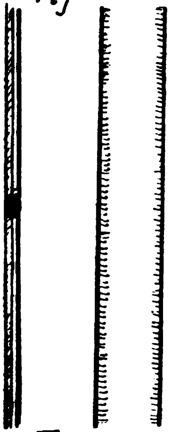


Fig. 4.

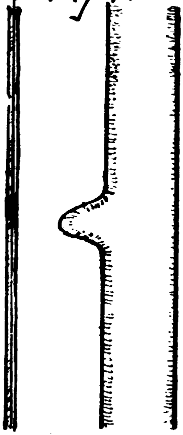


Fig. 5.

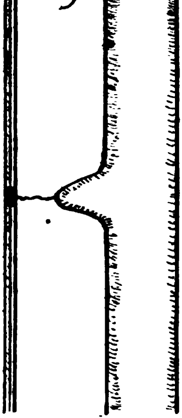


Fig. 6

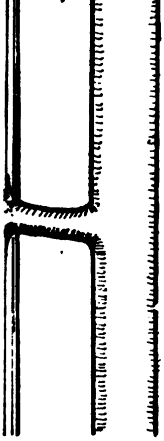


Fig. 7.

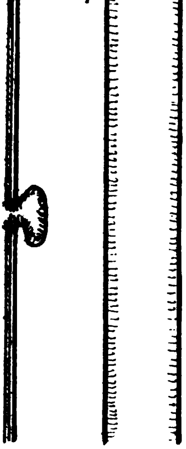


Fig. 8.

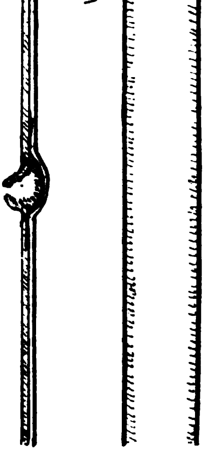
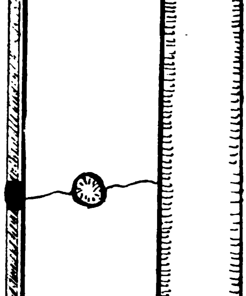


Fig. 9.



Other congenital anomalies are not rarely found in association with persistent omphalomesenteric remains, as, for example, transposition of the viscera, spina bifida, extroversion of the bladder, cleft palate, harelip, imperforate rectum and anus, patulous septum ventriculorum, etc. In my own case, as already mentioned, several congenital malformations were discovered. Cazin,[5] in a case of intestinal obstruction, was led by the existence of harelip in the patient to suspect a diverticulum as the cause of the strangulation, and found his suspicions verified at operation.

Fig. 10.

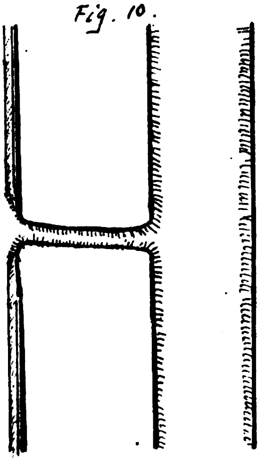


Fig. 11.

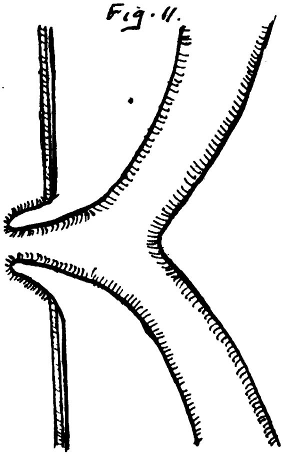


Fig. 12.

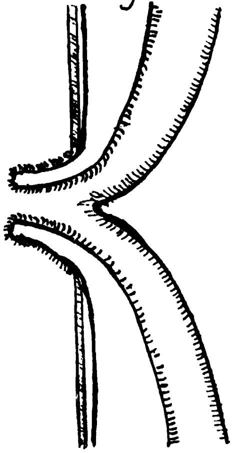
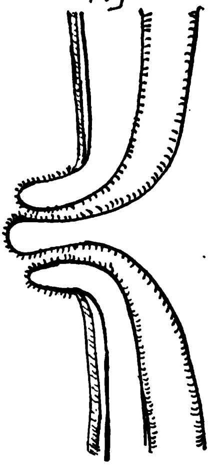


Fig. 13.



The most frequent condition met with in man is *persistence of the proximal part of the duct* as a blind pouch, known as *Meckel's diverticulum.* (See Fig. 4.) This curious process had been observed by numerous anatomists, among others by Lavater,[6] the first to see it, by Ruysch,[7] Haller,[8] Fabricius,[9] and Morgagni,[10] the last even seemed to have had a correct suspicion as to its origin; but the first one to prove that the diverticulum represented the original channel of communication between the yolk-sac and the intestine of the embryo was Meckel,[11] who published his observations in 1809. ["Meiner Meinung nach haben sie (die Divertikel) in der anfänglich Statt findenden Communication zwischen der Nabelblase und dem Darmkanale ihren Grund."]

The diverticulum springs usually from the ileum,—in 86 per cent of all cases, according to Lamb;[12] in a few instances it has arisen from the jejunum and the duodenum. It is generally attached to the convex border of the bowel, but sometimes it arises nearer the mesentery, and rarely, as in cases reported by Roth [13] and by Fitz,[14] it lies entirely within the mesentery. In the last class of cases the appearance is usually that of a duplication of the intestine, the diverticulum running parallel with the bowel in the same mesenteric fold.

The average length of the Meckel diverticulum is three inches, (seven and a half centimeters); sometimes it is only a nipple-like projection, and sometimes it attains, as in a case of Rokitansky's,[15] a length of ten inches. In the Museum of Morbid Anatomy of the University of Pennsylvania there are seven Meckel's diverticula, varying in length from four to seven centimeters. The shape of the diverticulum is usually like that of the finger of a glove; sometimes the process is conical, with the base at the intestine; occasionally it is bulbous, the end being expanded into an ampulla. This is the case in two of the seven in the University's collection. In another of the seven the end is knobbed on account of several small cystic projections and in one there is a small solid tumor at the end. Sometimes one or more small pouches project like accessory diverticula (*Nebendivertikel*) from the sides of the main process, as in a case of Neumann's.[16] In structure the diverticulum is identical with the intestine, with the coats of which its own coats are continuous. The mucous membrane may be smooth, or it may have, as one in the University Museum, well-marked valvulæ conniventes. The muscular coat may be deficient in places; this leads to hernial protrusions of the mucous membrane under the serous coat. It may also possess a Peyer's patch (Cazin[17]). Sometimes there is a valve at the junction of the diverticulum and the intestine; a short mesentery has also been observed, as in a case reported by Dr. Fussell. Phoebus[18] found a diverticulum with two openings into the intestine, one of which had a valve. In two cases the free end of the diverticulum contained an accessory pancreas (Zenker, Neumann), a condition, Leichtenstern[19] remarks, as interesting as it is difficult of explanation.

The identity of structure existing between the diverticulum and the bowel led Ahlfeld[20] to the opinion that the diverticulum was really not the remains of the omphalomesenteric duct, but represented a traction-pouch or traction-diverticulum of the bowel itself. In view, however, of the fact that remains of the duct that are not connected with the intestine have a structure almost identical with that of the latter, it is probable that the majority, if not all, of Meckel's diverticula are actually remains of the duct.

The relation of the diverticulum to the ileocecal valve is very variable. Meckel found the distance from the latter to the point of origin of the process to be four feet in adults and one foot in the newborn; Lamb states that practically in all cases the diverticulum arises from a point in the lower five feet of the ileum.

Cazin in one instance found it springing twenty lines from the caecum. In my own case, thecord arose at a point three centimeters above the ileocecal valve, and in the case of a child 11 weeks old, reported by Wernher,[21] a diverticulum two and a half inches long, connected with the umbilicus by a narrow canal, was attached one and a half inches above the caecum.

The diverticulum usually hangs free in the abdominal cavity, but it may be attached (*adherent diverticulum*), by means of a fibrous cord arising from its apex, to the anterior abdominal wall, generally near or at the umbilicus, or to the mesentery. (See Fig. 5.) This cord represents either the distal part of the duct, and, indeed, has at times a minute channel, or the obliterated omphalomesenteric vessels. The cord may be torn, and the ends hang free in the abdominal cavity, or they may form adhesions to another part of the bowel, the mesentery, or elsewhere.

The most frequent point of fixation of the diverticulum appears to be at the mesentery, this having been the case in twenty-four out of forty-six instances of intestinal obstruction from adherent diverticula collected by Neumann, and in the combined statistics of Cazin and Treves[22] such a mesenteric adhesion existed seventeen times in forty-two cases. The other cases tabulated by Neumann distribute themselves as follows: in five the adhesion was to the navel; in six to points on the abdominal wall at a distance from the umbilicus; in nine to other parts of the bowel or other organs; once, in the case reported by Jamain,[23] to an epiploic appendix; once to the vermiform process (Chiari[24]); once to the bladder (McGill[25]); in one case there were several adhesions, and in one there was a cord without a diverticulum. In the last case, reported in 1833 by Wagner,[26] the cord arose from the ileum and was inserted three inches above the ileocecal valve into the mesentery. It had caused a fatal strangulation.

Rarely the diverticulum is adherent by its free end without the interposition of a cord, as in the case of Hüber,[27] in which it was attached at the umbilicus.

One of the first instances of an adherent diverticulum, recorded in literature is that of Sandifort,[28] published with an excellent illustration in 1777. The anomaly was found by him in an infant that had died during delivery, and he speculates with much acumen on the possible effects of the adherent diverticulum had the infant survived its natal day.* Sometimes, as in one of Neumann's personal cases, the cord arises not from the apex but somewhere along the course of the diverticulum.

In rare cases the diverticulum finds its way into a hernial sac; it may also become invaginated into the bowel, a condition to which I shall again refer.

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* Appendix hæc ex intestino ileo enascebatur et singulari modo cum mesenterio cohærebat. Ex parte illa intestini, quæ mesenterio opposita ostio, in cæcum finem desinens, ejusdem circiter capacitatis, ejusdem crassitei, quoad tunicas, ac intestinum ipsum, facillime aerem admittens, illoque distentum, apice incurvatum. Ex illo apice filum enascebatur tenue, forte tamen, ad superiorem mesenterii partem tendens, illique diverticulum hoc adnectens. . . . Pluribus vero malis, si natalem diem supervixisset infans, ansam dare potuisse hanc præternaturalem conformationem, ex insigni intestinorum motilitate, et facile enascende constrictione, quisque concludet, præsertim cum in cadaveribus hominum, iliaca passione ereptorum, simile quid visum fuit. Caput X. De appendice ex intestino ileo enata.

The pathologic importance of Meckel's diverticulum is founded principally on two facts: first, that ulcerative lesions occurring in it are very prone to lead to *perforation*, and, secondly, that it is very apt to cause *strangulation of the bowel.*

Perforation may be due to the entrance of foreign bodies into the process. In a case observed by Körte,[29] a mass of cherry-stones, and in a case of Beale'[30] a cherry-stone, the coriaceous coverings of several orange-pips, and a concretion resembling those found in the intestines of horses, had caused perforation. If typhoid fever attacks the possessor of a diverticulum, the ulcerative process may, and probably nearly always does, extend into it, and then perforation is extremely likely to occur. I saw such a condition at an autopsy made last year; the specimen was exhibited before the Pathological Society by Dr. W. E. Hughes. Dr. Cattell, I believe, has also met with a case. Tuberculous ulcers may be found in the diverticulum, as in a specimen described by Fitz, which was the seat of three large ulcers.

Probably the most important morbid condition produced by the diverticulum is intestinal obstruction, the process coiling itself about a loop of bowel. This is especially apt to occur when the diverticulum has an expanded ampulla-shaped end, as that will prevent any knot that may be formed about a loop of bowel from being loosened. But strangulation by a free diverticulum is comparatively rare, for obvious reasons; it is only slightly more frequent than strangulation by the vermiform appendix. But when the diverticulum is attached by a band, as it often is, the chances for obstruction are greatly augmented. The band, as already stated, is most often affixed to the mesentery, and usually represents the obliterated vitelline vessels. A loop of bowel slips under the diverticulum and cord, and is constricted.

Remains of the vitelline vessels may also be found when there is no diverticulum. They present themselves in the form of bands or cords, usually solid, sometimes hollow, running from the anterior abdominal wall, especially the umbilical region, to the intestine or mesentery; at times both ends of the band are attached to the mesentery, or one to the mesentery and the other to the intestine.

Constricting bands are frequently interpreted as due to peritonitis, and such an explanation may be the proper one for some, as in a case that recently came under my observation, in which a cylindric cord between two and three inches long connected two loops of bowel, its point of insertion being, in each loop, at the base of a tuberculous ulcer. However, it is extremely likely that in many of the reported cases of intestinal obstruction by bands, the latter were the remains of the vitelline vessels.

An interesting case of this kind was reported to the Philadelphia Pathological Society by Dr. Roberts,[31] October 28, 1897. In a middle-aged man, who had died of intestinal obstruction, the autopsy revealed a loop of bowel encircled by a thin cord of tissue, looking like white string about the size used for tying grocers' parcels. The band or cord was thirteen centimeters long, and was attached at one end to the parietal peritoneum on the front of the abdominal wall and at the other to the mesentery above the upper portion of the constricted bowel. From another part of the bowel hung a pedunculated mass of tissue, four and a half centimeters long, which looked like several lymphatic glands held together by connective tissue. The thread-like band ran through a little opening in this appendage as a cord runs through a pulley. The appendage was attached to the intestine opposite the mesentery, but had no opening into it, as would be expected if it had been a Meckel's diverticulum. There was no other evidence of peritonitis. The vermiform appendix was normal.

Whatever the nature of the peculiar appendage may have been, there can, I think, be but little doubt as to the character of the long band; it clearly represented the obliterated omphalomesenteric vessels. I have already referred to Wagner's case, which closely resembles that of Dr. Roberts.

In a case of persistent omphalomesenteric duct, recorded by Deschin,[32] in a boy 5 months old, an additional cord was found arising at the same level as the duct, but from the mesenteric wall of bowel, and passing towards the mesentery. Diakonow considered this cord the obliterated omphalomesenteric vessels, undoubtedly the only proper view, and one which is put beyond question by the coexistence of another part of the omphalomesenteric structure. Had the duct, as might have been the case, been obliterated, the cord would perhaps have been looked upon as a stretched peritoneal adhesion. Neumann,[33] moreover, was able to demonstrate by dissection in two cases, a direct continuity between the cord attached to the diverticulum and the superior mesenteric artery. Fitz, in his excellent article, also emphazsized the fact, which I have stated above, that the majority of constricting bands causing intestinal obstruction are the remains of the omphalomesenteric vessels, and not the result of peritonitis.

Strangulation by diverticula is especially common early in life,—in twenty-four out of thirty-five cases collected by Neumann it occurred in persons under the age of 30. It is far more frequent in the male than in the female sex. Leichtenstern among forty-six cases found thirty-one men and eight women; in seven the sex was not given. The average age of these cases was 25 years.

Among the rarest consequences of diverticula is to be mentioned *intussusception.* This is produced by an inversion of the diverticulum into the bowel, the inverted process causing the part of the intestine from which it arises to slip into the part beyond. A case of this kind was reported a few weeks ago by von Stubenrauch[34] to the Congress of German Surgeons. The patient was a child. An operation was performed five days after the onset of symptoms, but gangrene of the entire invaginated portion had already set in, and led to a fatal termination. A specimen of an invagination due to an inverted diverticulum is also described by Treves,[35] from the Museum of Guy's Hospital.

Persistence of the Entire Duct.—Instead of only the proximal part remaining, the entire omphalomesenteric duct may persist as a tubular canal lined with columnar epithelium. (See Figs. 6 and 10.) In such cases a *fistula* results, and there is noticed at the navel, soon after the dropping off of the umbilical cord, a small reddish excrescence from which moisture escapes in the form of mucus, sometimes bile-stained; occasionally, and in some cases almost constantly, a little fecal matter is extruded (*fecal fistula*). The opening at the umbilicus is usually small, and when there is no fecal discharge, it is difficult to differentiate between patulous vitelline duct and patulous urachus. Roth[36] suggested that in such cases a little fecal matter might perhaps be obtained by means of a catheter.

The origin of the fistula is as follows: For some unexplained reason the omphalomesenteric duct persists and extends for a considerable distance into the umbilical cord. When the latter drops off, the duct which has formed adhesions to the edges of the umbilicus, is opened. The navel in these cases is usually very wide. Sometimes the mouth of the duct is closed for a time after birth, and only gives way on the occurrence of a violent straining effort, as during a spasm of whooping cough.

In addition to fecal fistula a persistent vitelline duct is capable of producing more serious conditions. The first of these that we shall consider is *prolapse of the canal.* (See Figs. 11 and 12.) This produces the formation at the umbilicus of a short, cylindric, reddish tumor, which is covered with mucous membrane and has an opening at its summit. The mucous membrane becomes continuous at the umbilical ring with the integument of the abdomen. A probe introduced into the opening may pass into the bowel, and may cause the escape of a little fluid or fecal matter. A prolapsed urachus, as Barth[37] says, might produce a prominence of similar aspect, but no case of this has been recorded.

Another consequence, conditioned on the one just described, is *prolapse of the bowel* itself. (See Fig. 13.) The child's straining and pressing forces the loop of gut to which the duct is attached into the latter and then gradually outward, in the form of a reddish, U-shaped or bicornate mass, sometimes sausage-shaped, and several inches in length; in one case, that of King,[38] the prolapse measured as much as a foot. The mass is covered with mucous membrane, which extends down into the two openings that are found one at each side of the summit, and at the base of the tumor is continuous with the skin of the abdomen.

The accompanying diagrams will serve to illustrate the conditions described. (See Figs. 11–13.)

Prolapse of the duct alone is very serious. Sauer[39] collected twenty-four cases, nineteen of which had proved fatal. One danger arises from the operative procedure that the physician, not realizing the true nature of the process, feels tempted to adopt when he sees the reddish prominence at the umbilicus,—namely, ablation. A reference to Fig. 11 will show that he thereby opens directly the abdominal cavity.

As for intestinal prolapse through a patulous duct, the accident, according to the most recent writer (Kö.te) has always proved fatal, the children dying rapidly with violent symptoms of intestinal obstruction. In one case reported by Barth, a very interesting condition had been superadded to the prolapse,—loops of bowel had been incarcerated between the serous covering of the duct and the serosa of the abdominal wall, after the manner of a hernia.

Persistence of the Distal Part of the Duct.—The umbilical part of the duct sometimes remains, and its persistence may give rise to two somewhat different conditions,—first, the part in the immediate vicinity of the umbilicus is not obliterated and is transformed into a *preperitoneal cyst*, lying just behind the abdominal wall. (See Fig. 7.) Such cysts are lined with mucous membrane, in which follicles of Lieberkühn may be found, and which is covered with columnar epithelium, and often bears goblet-cells. These cysts have been described by Roser,[40] Küstner,[41] and others. A fibrous cord may pass from the abdominal wall to the intestine. Secondly, the part of the duct in front of the abdominal wall remains, and is converted into a cyst, lying in front of the closed umbilicus. (See Fig. 8.) In the instance reported by Zumwinkel,[42] the only one of the kind on record, the cyst secreted a mucoid discharge, and after removal was found to have a lining of mucous membrane with cylindric epithelium, villi, and follicles of Lieberkühn; there were also a muscularis mucosæ and two layers of muscle. Solitary follicles were not present, nor do they seem to have been observed in any reported tumor of the umbilicus.

The cysts just described may become inverted, and give rise to the appearance at the umbilicus of a reddish tumor, covered with mucous membrane.

Another and very peculiar condition to be described is *persistence of the duct for a certain distance somewhere between the ends*, and the cystic dilatation of the vestigial part. (See Fig. 9) The cysts thus produced, the so-called *enterocystomata*, were first properly interpreted by Roth.[43] They are congenital, are fouud in the abdominal cavity, and in their walls represent more or less perfectly the structure of the intestinal canal. They may or may not communicate with the latter. The mucosa possesses villi and tubular glands, and is covered with columnar epithelium. Cysts anatomically similar may be produced, as Roth has shown, by constriction of a normally formed bowel through fetal peritonitis or through torsion of the mesentery; still others represent a rudimentary twin.

I now come to the last anomaly—namely, *persistence of the omphalomesenteric vessels.* Sufficient has been said about this under the head of Meckel's diverticulum, where the important part played by these remains in the production of internal strangulation was pointed out. As far as the cord found in my case is concerned, I am inclined to look upon it as the remains of the omphalomesenteric vessels, rather than as an obliterated duct, first, because of its tenuity; secondly, because of the absence of even the slightest indication of a diverticulum; thirdly, because of its insertion rather near to the mesentery; and, fourthly, because of the close proximity of its intestinal end to the ileocecal valve, diverticula, and ducts generally, though not always, arising higher up, while the insertion of vessels is more erratic.

CONCLUSIONS.

1. Remains of the omphalomesenteric structures are not rarely found.
2. Persistence of these structures may affect the proximal part of the duct (Meckel's diverticulum), the distal part of the duct, the entire duct, the more central parts, or the blood-vessels.
3. The dangers from Meckel's diverticulum are perforation from ulcerative processes and foreign bodies, strangulation, and intussusception of the intestines.
4. Strangulation is most apt to occur when the diverticulum is adherent. It is most common in the male sex and in early life.
5. Persistence of the entire duct may lead to fecal fistula, to prolapse of the duct, or to prolapse of the bowel with strangulation.
6. Persistence of the vessels, with or without persistence of part of the duct, very often leads to strangulation of the intestines.

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EXPLANATION OF ILLUSTRATIONS.

Figs. 3 to 13 illustrate formation of omphalomesenteric anomalies, and are explained in the text. Fig. 3 illustrates the normal condition. The two lines with oblique shading between them, represent the abdominal wall; the heavy line, peritoneum; the vertical shading, mucous membrane.

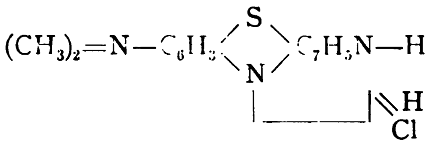
May 12, 1898.

Two New Methods of Staining the Axis-Cylinders of Nerves in the Fresh State. Some Microchemic Reactions of Toluidin Blue.

H. F. HARRIS, M. D.

Toluidin blue is chemically nearly related to both thionin and methylene blue, and, as might have been expected, bears a very close relation to these substances in its microchemic reactions.

It is chemically the hydrochloride of dimethyltoluthionin and has the formula:



The commercial article, like methylene blue, often occurs in combination with the chloride of zinc. Further than the above facts I have been unable to obtain information concerning either the physical properties or chemistry of this substance.

Toluidin blue occurs as a crystaline powder, of a dark slaty blue color, and is readily soluble in water and alcohol,—to both of which it imparts a dark blue color. I have made no attempt to study the chemistry of the compound in a comprehensive way, but for the purpose of gaining a general idea of its chemic peculiarities, I have noted its behavior in the presence of several easily decomposable reagents. It remains unchanged in the presence of hydrogen dioxide, and when heated with the higher oxides of mercury, but is precipitated by potassium permanganate on the application of heat; when a solution of potassium bichromate is added to it, there occurs a brownish precipitate. Its solutions are decolorized by phenylhydrazin when heated, or in the cold after the addition of an alkali. Chloral hydrate and pyrogallol both decolorize solutions of the dye when heated with it in alkaline solutions. It is likewise, under the same circumstances, entirely decolorized by glucose, for which it may be used as test. All of these decolorized solutions will more or less regain their color by agitating them for a time in the presence of oxygen. It is precipitated in the cold by the addition of silicate of sodium, ammonium molydbate, the picrates, the salts of benzidine, acid aniline dyes, the bichloride of mercury, and by both the ferro- and ferri-cyanides of potassium.

A search into the literature has only brought to light a few cursory notices of its microchemic properties. Hoyer[1] seems to have been the first to take advantage of its staining properties and has mentioned in his very able and thorough article that aqueous solutions of this substance give excellent results as a stain for mucin; he also calls attention to the fact that it gives polychromatic effects. It was later highly recommended in combination with eosin as a stain for ganglion cells by Mann.[2] Its power of staining chromatin is referred to in that most excellent book "The Microtomist Vade-Mecum" by Lee. I[3] recently called attention to the fact that it gives very beautiful results as a stain for the amœba coli, and that it also stains malarial organisms well. In this communication it is my purpose to point out some further uses to which this dye may be put as a stain for tissues, and which together with the reaction just referred to, should place it among the most prominent of the staining reagents.

Aqueous solutions of the dye should alone be employed for staining purposes. Alcoholic solutions produce very faint results, and, furthermore, the staining is diffuse, and does not exhibit the marked electivities which are so abundantly observed in tissues when the watery solutions are used. The strengths of the solutions employed have varied from 0.1 per cent to 3 per cent—the latter being a saturated solution; there is no advantage in using strong solutions, as the results were always the same, no matter what the degree of concentration. Profiting by our experiences with other stains, I have dissolved various substances in the water before adding the dye, such as aniline oil, carbolic acid and formalin; I have also added alkalies and acids—among the former borax, lithium carbonate, and carbonate and hydroxide of sodium, and of the latter hydrochloric, nitric, sulphuric, acetic, formic and oxalic acids. The solutions in aniline water exactly resembled in their staining properties those made with water alone, but with the others, more or less marked differences were noted in each instance. When simple watery solutions are used the entire tissue is stained a uniform blue, but when differentiated by alcohol or other means, it is found that the stain adheres much more tenaciously to the chromatin of the nuclei and to the protoplasm of certain cells than it does to the remaining part of the tissues; it follows that by proper care these objects can be brilliantly stained, while the surrounding tissues are practically colorless. When acid solutions are used, chromatin and the protoplasm of basophilic cells are alone stained; the staining is not so rapid as when other solutions are used. If the stain be made alkaline the nuclei and the protoplasm of all cells, elastic, collagenous and muscular tissues are stained intensely; the staining is very rapid. The formalin solutions give a stain much resembling those produced by acid solutions, while on the other hand the carbolic acid solutions give effects which are not unlike those got with alkaline solutions. The carbolic—acid—toluidin blue is much to be recommended, as it acts quickly, stains very deeply and will bear any amount of washing with alcohol; it is further a desirable combination inasmuch as its reaction is, if not acid, certainly neutral.

Stained sections were washed in solutions of the substances above mentioned as precipitating toluidin blue, and it was found that of these the molybdate of ammonium and the ferro- and ferricyanides of potassium rapidly and firmly mordant the stain in the tissues.

After staining the different elements in the tissues may be differentiated by means of alcohol alone, or alcohol acidulated with any of the vegetated or so-called mineral acids,—the strengths used being for ordinary purposes, from 1-2 per cent of the acid added to either pure or dilute alcohol. Watery solutions of the acids may also be employed. Aniline oil alone, the various mixtures with xylol or Unna's alum-aniline may be used for the same purpose.

Solutions of tannic acid, creosol and Unna's ether-glycerine mixture all act well. Perhaps, however, styron in ordinary work is the best differentiating agent. If the tissues are mordanted, differentiation can only be secured by means of the acid mixtures, and preferably, those made with mineral acids. If the mordant be allowed to act longer than one or two seconds, even the acid solutions do not remove the excess of stain in a satisfactory manner. I find, however, that after any degree of mordanting the excess of stain is slowly but in a very selective manner removed by watery solutions of tannic acid; I always use a saturated solution.

The above remarks apply only to the staining of tissues which have been fixed, but, in addition, I have recently discovered that fresh tissues may be also stained with this dye. I think investigation will show that there is no reaction which can be got with methylene blue that cannot be equally as well obtained with toluidin blue. Like methylene blue it stains the cement substance between epithelial cells, and has also the important property of staining the axis-cylinders of nerves in the fresh state. Solutions of the dye may be injected into the living animal, or pieces of the fresh tissue may be placed in solutions of the stain. For staining nerves weak solutions are much to be preferred to strong ones,—my best results having been obtained with solutions of a strength of from 1 to 1000 to 1 to 4000.

For staining pieces of tissue I find that a solution made in the following way may be recommended: Toluidin blue in physiologic salt solution $\frac{1}{1000}$, 2 parts; ammonium chloride in water, 0.25 per cent. 1 part; egg albumen, 1 part.

The solution should be freshly prepared. The tissue should never be covered by the solution, but from time to time only enough added to keep it moist. The staining is generally complete in from one-half to an hour. I think it advisable to keep the tissue in a moist chamber at a temperature of about 37° C. while staining. After the stain attains its maximum of intensity it slowly fades, so that if it is desirable to make a permanent preparation it is necessary to fix the stain at this stage. Any of the methods which have been used for the purpose of fixing the methylene blue stain can be here also employed, and the technique is the same in both cases. I have also devised a method which may be used for fixing the stains of either the toluidin blue or methylene blue, and which, I think, is of somewhat easier application if it is superior in no other particular. As soon as the desired degree of intensity of the stain has been secured the speciman is rinsed in water and transferred to a saturated solution of either ferro- or ferri-cyanide of potassium which has been cooled to within a few degrees of zero. A trace of osmic oxide added to the mordanting solutions prevents the slight macerating effect which is otherwise observed. At this temperature the object should remain until it is removed at the end of from three to twenty-four hours. It is then washed for an hour in distilled water, and is dehydrated in absolute alcohol, which should also be kept at a low temperature. After dehydration the specimen is cleared in xylol or cedar oil and is embedded in paraffin.

When the stain is injected into the living animal, I commonly use a solution made by adding one part of the dye to 1000 parts of physiologic salt solution. In about an hour after the injection, the animal is killed and small pieces of the tissue cut out and exposed to the air for ten or fifteen minutes, at the end of which time the nerves will be stained in most instances. During the period of exposure the tissue should be kept moist with physiologic salt solution. The after treatment is the same as when the tissues have been stained directly in the solution of the dye. Sensory nerves, as is the case with methylene blue, are stained before the motor nerves.

It will be observed that the technique of staining fresh tissues with toluidin blue is exactly the same as that which is employed when methylene blue is used for a like purpose, and as these methods are so well known, I do not deem it necessary to discuss the details minutely.

My experience with this stain is not yet sufficiently great to admit of any positive statement as to its merit as compared with methylene blue, but it nevertheless exhibits peculiarities which lead me to think that it may possibly possess some point of superiority. In the first place I have never yet experienced difficulty in obtaining the reaction, and I have used stains from two different sources. Furthermore, the reactions have been quite as satisfactory when a stain which had been exposed to the air for four years was used as when a perfectly fresh article was employed. The tissues for the most part are stained blue, while the axis-cylinders of the nerves assume a purple color, which makes them stand out quite prominently from the surrounding blue field. This purple color of the nerves is preserved and even accentuated when picrate of ammonium is used for fixing the stain, and the color is well preserved when either the ferro- or ferri-cyanide of potassium is employed for a like purpose, but they become blue if the fixation is accomplished by molybdate of ammonium. As to its permanence, I can as yet say nothing.

My success with toluidin blue led me to attempt the staining of fresh nerves by means of thionin, and the results obtained were scarcely inferior to those which were got by means of the former reagent. Indeed it may be said that the stain with thionin is superior to either that of methylene blue or toluidin blue in the fact that the surrounding tissues are not stained so deeply. The axis-cylinders are stained of a purple color, but not so intensely as by the toluidin blue. The technique of staining is in every way the same as when toluidin blue or methylene blue is used, and the stain may be fixed by the same methods.

In addition to the above I desire to call attention to some microchemic reactions of toluidin blue which I think will prove of general value to microscopists. We are all acquainted with the fact that gentian violet, methyl violet and methyl green have the property of staining amyloid material red, but it is equally well known that, unfortunately, the stain cannot be mounted in balsam, and that it rapidly fades. With the object of obtaining, if possible, a permanent differential stain for amyloid, the following method has been devised. Sections of material hardened in alcohol are stained for from three to twenty-four hours in carbol-toluidin blue, are rinsed with water and the stain is mordanted for one or two seconds in solutions of ferro- or ferri-cyanide of potassium or molybdate of ammonium; the mordant is washed off with water and the section differentiated in acid alcohol. For this purpose a 1 per cent solution of either oxalic or hydrochloric acid is probably to be preferred; the addition of as much as 50 per cent of water to the alcohol does not interfere with the result. After differentiation the specimen is dehydrated, cleared and mounted. Before dehydrating the section, the stain should be further mordanted by any of the above-mentioned mordants, in case the specimen is to be much exposed to strong lights, as without this the stain fades considerably after several days—especially if it be exposed to the direct rays of the sun. The amyloid material is stained red while the remaining histologic structures of the tissues are for the most part stained in varying shades of blue, the nuclei being especially well brought out. The exception to this is that fibrous structures are often stained of a more or less reddish color, but never so deeply as the amyloid. This does not lead to confusion, as in addition to the deeper shade of red which the amyloid exhibits, its homogeneous character serves to distinguish it.

Attempts to obtain this reaction by substituting methylene blue and thionin were unsuccessful, but some color differentiations in shades of blue were secured.

Toluidin blue is also a specific stain for the medullary sheaths of nerves which have been hardened in the salts of chromium. As is the case when using Wiegert's hæmatoxylin process it is necessary for the material to remain sufficiently long in the chromate solution to have acquired a brown color. The tissue is washed, dehydrated and embedded in the usual way. Sections are stained for several hours in an alkaline solution of toluidin blue, and are then differentiated in a saturated aqueous solution of tannic acid. The medullary sheaths are stained a dark slaty-blue color, while the surrounding tissues are greenish; nuclei are beautifully brought out. The stain may be also differentiated by a 1 per cent alcoholic solution of oxalic acid; in this case the fibrous structures are stained red, the medullary sheaths appear faintly yellow, while the axis-cylinders are dark blue in color. The result is not unlike that which is seen after staining by Van Gieson's method. While any alkaline solution of the stain may be used a 1 per cent borax solution in water, to which 1 per cent of the dye is added, is especially to be recommended.

For the purpose of studying either nuclei or the basophilic granules which are present in many cells, I know of no reagent which is superior to toluidin blue. The tissues can be hardened in any of the ordinary fixing solutions, but by far the best results are secured after the use of mercury bichloride. The traces of this substance which cling to the tissues undoubtedly play the part of mordant, and consequently the staining is rapid and the coloration brilliant. Sections may be stained by any of the solutions mentioned in the early part of this paper, but on account of the rapidity of its action and the tenacity with which the resulting stain adheres to the tissues, the carbolic acid water solution is to be preferred. Sections stained in this medium for several minutes, and subsequently differentiated in alcohol, show nuclei and the protoplasm of basophilic cells stained intensely blue, while the protoplasm of the mast cells is of a very dark red color; connective tissue and muscle are stained light blue. A very fine double stain is secured if the sections are stained with eosin or benzopurpurin either before or after the toluidin blue is used. If the stain be differentiated in water or alcohol, to which 1 per cent of some acid has been added, the protoplasm of the mast-cells stain a bright red color, while the connective tissues and muscles are in a large measure deprived of their color; if oxalic acid be used in this way, the connective tissues stain a pale reddish color. If the tissues are fixed in Flemming's or Hermann's solution the mast-cells are stained a dark blue color, while nuclei and the protoplasm of plasma cells assume a greenish hue. Tannic acid solutions quickly dissolve the stain from the protoplasmic and intercellular substances, causing them to assume a yellow color, while the nuclei remain intensely stained; this method affords a very clear view of the relation of the nuclei to the protoplasm of cells. Beautiful differentiation of all histologic elements are secured by using any toluidin blue solution followed by styron; this substance abstracts the stain much more slowly from involuntary muscle than from the connective tissues, and hence the former is always considerably darker than the latter in sections prepared in this way.

Bacteria are well stained by toluidin blue, especially in alkaline and carbolic-acid-water solutions. The stain may be mordanted by the reagents which effect a like result in animal tissues.

In concluding the subject I wish again to call attention to the beautiful results which are obtained by staining ganglion cells with toluidin blue; the chromophilic substance of these cells is much more intensely stained in this way than by either methylene blue or thionin. Any degree of intensity of coloration can be easily secured by after treatment with any one of the above mentioned differentiating agents. In this connection it may be noted that those basophilic but otherwise hyaline-like bodies, so frequently encountered in the central nervous system when degenerative changes are in progress, are deeply stained by toluidin blue.

In summing up the microchemic properties of toluidin blue as compared with other members of the group to which it belongs, perhaps its most striking peculiarity is that the depth of the coloration which it produces is more intense than that which can be secured by means of methylene blue or thionin, and it exhibits a much greater tendency to produce polychromic effects. Furthermore it is not washed out by acids and acid dyes so readily as are either of these reagents. Its property of being easily mordanted—which it shares with methylene blue, and to a limited extent with thionin—is also of much value, as we are enabled in this way to fix any desired degree of intensity of the stain; nor is this the only value of the procedure, as after the dye has been mordanted it shows much less tendency to fade. This seems to be in consequence of a general law in connection with stains, namely, that they fade directly in proportion to the ease with which they may be washed from the tissues. As to the stability of toluidin blue, I can only say that I have never been able to observe the slightest difference between the staining properties of old and new solutions; a substance, however, which is chemically so intimately related to methylene blue should be looked upon as being probably easily decomposable, until positive proof to the contrary is brought forward. It is possible that its polychromic properties are due to the presence of impurities, such as methylene red; I am, however, rather disposed to believe that these effects are the result of differences in the chemic composition of the tissues. Toluidin blue shows on the one hand less tendency to stain protoplasm and connective tissue structures than methylene blue, while on the other it is not such a pure nuclear stain as thionin; it may, therefore, be regarded as being intermediate between these substances in its microchemic properties.

Although I have been unable to test the matter, it is probable that thionin blue, on account of its near relationship to the stains referred to in this paper, will be found to give similar reactions with tissues.

(1) Hoyer, Arch. f. Mik. Anat., XXXVI, 1890. (2) Mann, Zeit. f. w. Mik., XI, 4, 1894. P. 489. (3) Harris, Amer. Jour. Med. Sciences, April, 1898.

Discussion.

Dr. Spiller remarked that in his experience thionin had given as good results in staining the nerve cells as toluidin blue, and inquired whether axis cylinders could be stained in teased preparations by the latter.

Dr. Harris answered in the affirmative, but stated that the preparations were prone to fade.

May 12, 1898.

Nucleolar Degeneration of the Ganglion Cells.

From the Pepper Laboratory of Clinical Medicine.

JOSEPH SAILER, M. D.

The ganglion cells of the central nervous system are exceedingly irregular in shape, due to the projection from them of a various number of protoplasmic elongations or processes, which subdivide and branch, forming treelike outgrowths. A single process of different structure, the neuraxon, also proceeds from each cell, giving off from time to time, slender branches at right angles, the so-called collaterals. The neuraxon finally terminates in a tuft of fibres that surround another ganglion cell without touching it, or, if it projects into the peripheral nervous system, in the end plate of muscle, or in a sensory corpuscle of the skin. Nissl has divided the ganglion cells in the somatochromes and karyochromes according as the cytoplasm contains granules taking the basic anilin dyes or not. In the former, after suitable fixation, a number of basophilic masses, apparently composed of aggregations of small granules, are found in the cytoplasm. These are usually grouped more or less concentrically about the nucleus, although Lenhossek denies this, and extend as spindles into the protoplasmic processes. The arrangement is by no means uniform in the various species of animals. The concentric arrangement does not occur in the ganglion cells of the anterior cornua of the spinal cords of cattle, and these were the cells chiefly studied by Lenhossek. In the spinal cord of the horse, on the other hand, it seems to exist in the majority of cells, and it is often quite distinct in human spinal cords.

The nucleus is a round body, surrounded by a membrane that sometimes stains deeply, and, under normal conditions, is in or near the centre of the cell. It contains, in adult tissue, a very small amount of chromatin arranged in the usual reticular manner, and a deeply staining polychromophilic nucleolus, and often one or two accessory nucleoli (Koelliker). The latter stain in a somewhat different manner, that is to say, with methylen blue they are green, and in sections stained with Rosin's solution they have a reddish tinge. In specimens stained with hematoxylin, karyosomes may sometimes be observed. Two true nucleoli are sometimes found in the cells of other tissues (Wilson), and have been described in nerve cells by Ranvier, and pictured by several authors without description. The extraordinary feature of the nuclei of the ganglion cells is the fact that the chromatin, excepting in those of the granular layer of the cerebellum, does not stain with basic anilin dyes. A peculiar feature that I have observed in the cells of amphibia (Amphiuma) is that the so-called sensory cells or karyochromes, without chromophilic granules in the cytoplasm, have in the nucleus a large amount of chromatin that takes the basic anilin dyes. The motor cells, on the other hand with a dense mass of chromophilic granules in the narrow strip of the cytoplasm, have none in the nucleus, which has the appearance of a huge vacuole. The nucleolus is a small spherical body, 3 to 7 μ in diameter, that, according to Babes, contains an achromatic endonuclear substance, which is surrounded by chromatic and metachromatic granules, the latter probably the accessory nuclei already mentioned. It is usually placed in the centre of the nucleus, and almost invariably may be seen to contain one or two vacuoles that are deeply placed in its substance and do not cause any alteration in its outline. De Quervain believes that these are not vacuoles in the true sense of the term, but small highly refractive granules. Often small aggregations of chromatin, slightly darker than the rest of the nucleus, may be seen at its periphery, forming slight projections from its surface; these are found both in normal and pathological cells. Occasionally there is a ring of minute granules around the edge of the nucleolus, and in certain cases little rays may be observed projecting from these into the substance of the nucleus. This is possibly an artefact, although a similar appearance has been noted in the nerve cells of some of the invertebrates. Berger has recently described a small ductlike body that extends inward from the nuclear membrane, and divides into three branches, all of which point towards and partially enclose the nucleolus. This, however, has only been found in degenerated cells, but he holds that it is a normal structure rendered visible by the degenerative process. The post-mortem changes that occur in the ganglion cells have been carefully studied by Barbacci and Campacci; according to them the nucleolus is one of the last structures in the cell to be affected. It becomes irregular in outline, loses its tinctability, and may be found either at the periphery of the nucleus or entirely in the cytoplasm. Finally it breaks into fragments and disappears. This resistive power of the nucleolus renders it especially valuable as a criterion of pathological changes, and it seems to be accepted by neuropathologists that no normal cell possesses an atypical nucleolus.

The pathological alterations consist, first, of swelling with a relative decrease of the staining power; this is not necessarily a destructive change, for, as Goldcheisder and Flatau have shown, the cell may still return to its normal condition. In other cells that are slightly degenerated, particularly in some that I observed in a spinal cord just above the upper level of a spinal glioma, the nucleoli are slightly swollen, and there are present in the nucleus one or more large irregular masses of chromatin that stain with the basic anilin dyes, and may represent nucleolar fragmentation, although I am inclined to regard them as modified karyosomes with altered staining reactions.

Secondly, a more distinct alteration is the multiplication of the vacuoles; as these increase, the nucleolus loses its circular outline and appears as a mass of small globules; a condition that Berger has aptly compared to the morula stage of the ovum. Corresponding changes in the nucleus are displacement, even until it projects a considerable distance from the side of the cell; irregular thickening of the nuclear membrane, or else its complete disappearance; the development or increase of the staining power of the chromatin, and sometimes its accumulation as an irregular homogeneous mass about the nucleolus. This perinuclear accumulation of chromatic substance has been particularly figured by Babes in connection with leprosy, and I have observed it in the cells of the spinal cord of a horse that died of a disease known among veterinarians as azoturia. The vacuolization of the nucleolus is probably not a mere increase in its achromatic substance. I am rather inclined to believe, although I can base my opinion on nothing better than appearances, that there is an actual imbibition of liquid from the karyoplasm. In favor of this view is the fact that the nucleus alters its staining power distinctly, and that the vacuoles no longer show the clear, sharp outline and high refractive index characteristic of those seen in the healthy cells. As this change progresses, the nucleolus continues to swell, its outline becomes indistinct, and finally it is impossible to be certain whether it exists at all, in other words, it seems to be dissolved in the cell, a process that may perhaps be termed nucleolilysis. I have particularly observed this series of changes in the spinal cord of a criminal, in whose brain marked pathological changes were present. The cells of the column of Clark are particularly affected, indicating their tendency to degenerate easily in general conditions, a tendency to which Babes has called attention. They are swollen, pale or dark, their substance is homogeneous, the nuclei are often displaced toward the periphery, in some cases projecting from the cell, and in the majority, nucleolar changes can be distinctly observed. Finally the existence of two typical nuclei in one nucleus may be a pathological alteration, although I am not sure of this fact. I have observed this condition in the karyosomatic cells of the cortex, and in the cells of Purkinje of the cerebellum of a guinea-pig that was killed with tetanus toxin. Two nucleoli were frequently found, and, in a few instances, three or four could be observed. These are not chromatin knots; in the first place the section is stained with methylene blue, a dye that does not stain the chromatin of the nucleus, and in the second place, a good illumination and high powers reveal very distinctly the presence of accessory nucleoli. In all other respects the cells appear to be perfectly normal. Exactly this condition is pictured by Parascandolo in a recent work upon the changes in the cells of the central nervous system as a result of traumatic shock, and in 2 of the 31 figures, in which this condition is represented, the cells are extremely degenerated. Such cells have also been pictured by Held, but I have been unable to secure the original article. It is not impossible that these double nucleoli represent an abortive effort at cellular proliferation, the result of some abnormal stimulus acting upon the cells, although in the normal karyokinetic process, and also in those abnormal forms described by Galleotti and Stroebe in cancer cells and in lesions of the epithelium of salamanders kept in an unfavorable environment, the nucleolus disappears during division. It appears, therefore, that the nucleolus is usually affected late in the process of cellular disease, and that profound changes in its structure are invariably associated with the death of the cell. It seems reasonable, therefore, to conclude that it possesses some nutritive function, but whether this is trophic or secretory, it is of course impossible to determine. In studying the series of changes here described, I have noted that the nervous system of any one animal rarely exhibited more than one form of degeneration, and it is possible that the nucleolus reacts in a definite manner to different destructive agencies. My material, however, has not been sufficiently abundant to enable me to determine this point with any certainty.

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Heimann (Virch. Arch. 152, Heft 2) has recently studied very thoroughly the structure of the cells of the spinal ganglion. He states that from one to five nucleoli may be found in a single cell. From his remarkably beautiful illustrations, I am inclined to suspect that he has confused nucleoli and accessory nucleoli, and perhaps also karyosomes. (See Figs. 7, 10, 19, 21 and 22.)

April 14, 1898.

Miliary Carcinomatosis of the Peritoneum with Microscopic Slides Showing So-called Microorganisms of Cancer.

H. A. HARE, M. D., and H. F. HARRIS, M. D.

CLINICAL REPORT BY DR. HARE.

F. M., the patient, a woman of 55 years, a widow, was admitted to the Jefferson Hospital in January, 1898. Her mother and one sister died from some uterine trouble, her father of some disease of the liver. There is no history of tuberculosis. The patient has had the usual diseases of childhood. She had enteric fever at twenty years, which left her with a milk leg and a chronic ulcer that discharged pus for twelve years. Her menstrual life has been normal, she married at 25 years, had one child (birth normal), no miscarriages, and reached the menopause four years ago. She was operated on in this hospital, April 18, 1896; a double ovariotomy was done for cysts. She was discharged as cured, but ever since the operation she has had paroxysmal sharp shooting pains in the left hypochondriac region over the line of incision.

Three weeks before admission, following a severe cold, she began to have on exertion dyspnœa which became quite marked; when admitted she had orthopnœa. She had gradually lost strength and was scarcely able to walk. The bowels were costive.

The urine was neutral in reaction, dark amber in color, the sp. gr. was 1030, it contained no sugar by Fehling's or the bismuth test, and no albumen by the heat and nitric acid test; there were Urea gr. ix to f℥i.

On January 31 I aspirated the right pleural cavity, removing one pint of bloody fluid, and on February 3 I again aspirated the right pleural cavity, removing three pints of bloody fluid.

On February 8, in consultation with Dr. Montgomery, the abdomen was found to be enlarged and fluctuation was present.

Urine, 17½ oz. in twenty-four hours. Sp. gr. 1021. Reaction, acid. No albumen or sugar. Urea, 6 gr. to f℥i.

On February 11, she was operated upon by Prof. Montgomery; an incision was made in the right hypogastric region and four quarts of fluid removed, which was clear and straw-colored.

On February 19, the right pleural cavity was again aspirated and and 46 f℥ of dark, bloody fluid removed.

Urine examination, February, 23, 1898. Sp. gr., 1025. Reaction, faintly acid. Moderate amount of albumen. No sugar. Blood and pus in considerable quantity. Squamous epithelial cells.

On February 27, I again aspirated the right pleural, drawing off 46 f℥ of a brownish chocolate-colored fluid, which on standing deposited a slight reddish sediment. A microscopic examination revealed many red and white blood cells, the latter being mostly polynuclear. The sediment was composed mostly of red cells, a few leucocytes and fibrin shreds. A bacteriological examination failed to demonstate the presence of any micro-organism.

On March 7, 1898, the right pleura was again aspirated and 47 f℥ removed, resembling in appearance that of previous aspirations.

The fluid slowly reaccumulated and the patient grew gradually weaker. She declined further aspirations, and died on March 18.

Pathological report by Dr. Harris: The necropsy was made in the presence of relatives of the deceased and a thorough examination was not permitted. None of the organs were allowed to be taken out.

The post-mortem rigidity was slight. The body was well nourished, and a large amount of subcutaneous fat was present. The pleuræ contained large amounts of blood-stained serous fluid pushing the lungs upward and backward. There were also marked pleural adhesions, very dense in character, toward the apices; they were quite symmetrical.

The peritoneal coating of the abdominal viscera was everywhere studded with small nodules which never extended into the substance of the various organs. These nodules were wart-like bodies, some of which were so small that they were barely visible to the naked eye; the largest were but little over .25 c. m. in diameter and elevated to 4 to 5 m. m. above the level of the peritoneum. The top of each of these papillary outgrowths was whiter than the surrounding tissue and many of them were fissured on the surface, resembling ordinary warts. In a number of places the masses had run together as agminated masses over areas .5 to 1 c. m. wide and 2 to 5 c. m. long. These grouped masses of papillary growths had, in the more crowded portions, the appearance of being confluent. A few of the masses showed delicate blood vessels coursing over the surface as an arborescent network. The mesentery contained but few papillary masses on its surface.

Pieces of tissue taken from the intestinal wall, from the superficial portions of the liver and spleen and from the omentum, were immediately fixed in Heidenhain's $Hg.Cl_2$ solution, and were subsequently embedded in paraffin. Sections were stained with Unna's acid orcein and in his alkaline methylene blue followed by neutral orcein; sections were also stained with carbol-toluidin blue followed by acid alcohol, and others counter-stained by orcein and eosin. Stains were also made with hematoxylin and eosin, and carmalum and picric acid.

On microscopic examination it is seen that the small tumors above referred to are the result of proliferation of the tissues of the subperitoneal coat of the intestines, or of the capsules of the organs examined, with the addition of certain elements which have the appearance of masses of very atypical cylindrical epithelium. The process has shown no tendency to penetrate into the substance of the organs, or to infiltrate deeper into the intestinal wall. The stroma of the tumors is made up of a reticular network of somewhat modified collagenous tissue, the change being a diminished tendency on the part of the tissue to take up acid dyes and a much increased affinity for basic ones. Numerous blood-vessels are present within the stroma, but they can scarcely be considered as having distinct walls. The flattened endothelial plates which line these vessels seem to be directly in contact with the surrounding fibrous structures without the interposition of muscle fibres. The cellular elements within the stroma are moderately numerous, their number being generally greater in the smaller tumors; the cells are frequently collected in small groups and these generally lie along the free surface of the tumors. Scattered through the tissues are irregularly shaped cells, with large rounded or spindle-shaped nuclei and with acidophilic protoplasm; the nuclei sometimes stain very faintly, and in some situations not at all. These are evidently connective tissue cells. The small collections of cells above referred to consist principally of lymphoid cells, but there are also many polynuclear leucocytes and some plasma cells. There are no mastcells.

Between the bundles of collagenous tissue there are numerous irregularly rounded openings varying in diameter from 60 to 200 μ. The tissue forming the walls of these openings is for the most part bare, but in some cases is lined by cells which have the appearance of atypical columnar epithelium; these are often piled upon each other three or four rows deep. The contents of these alveoli consists of extremely irregular masses of cells which partake of the peculiarities just referred to; these masses never entirely fill the openings,—oftentimes occupying only a fraction of the space contained within them. The cells are often so closely crowded together that it is impossible to clearly make out their boundaries. Their protoplasm is faintly granular and stains with both acid and basic aniline dyes, but shows more affinity for the latter; the nuclei are large and vesicular in character and exhibit a decided loss of power to take nuclear stains. The cells have a diameter of from 8 to 12.μ Both within these supposed epithelial masses and within the stroma of the tumors there are bodies which present the morphological and staining peculiarities of the supposed parasites of cancer to which Sanfelice and Roncali have within recent years so often called attention. These bodies, which the above named observers regard as blastomycetes, were first observed after staining in toluidin blue followed by an aqueous solution of oxalic acid; the organisms were stained of a dark blue color, while the encapsulating substance assumed a pale red tint. Specimens were also stained by the gentian violet method of Sanfelice and the results demonstrated beyond doubt that the bodies are identical with the ones described by this author. These bodies vary in size from 9 to 28,μ and are almost invariably encapsulated. The capsules are almost always much larger than the enclosed organism, and vary from 17 to 40μ in diameter. The organisms are generally round or oval in form, but sometimes there are numerous rounded projections from their sides. Under a high power they are faintly granular, and their peripheral portions are usually more densely stained than their centers.

DISCUSSION.

DR. COPLIN.—These growths to my mind represent the mural implantation incident to invasion of the peritoneum by an ovarian papilloma; they are not metastases by the blood stream or lymph circulation in the ordinary acceptation of the term. Structurally the sections do not represent the typical cylindric celled epithelioma which at the first glance one is inclined to believe them to be. The so-called cancer parasites show very well in these sections, it is indeed in such cases that they are best seen. I have seen similar cases in which, however, unfortunately the histology was not so carefully worked up as in this case.

Dr. Sailer referred to a case, recently reported by Jürgens, in which multiple tumors were found in the brain, heart and kidneys of a child that died at the age of six months. Parasites were demonstrated in smear preparations from the tumors in the heart and brain. Rabbits were inoculated with an emulsion of the brain substance, and in one that died, after eight days, some parasitic growths were found in the heart.

May 26, 1898.

Specimens of Cardiac Lesions.

H. A. HARE, M. D.

The first specimen is that of a case of mitral stenosis. The button-hole orifice is very well marked. The case is of greater interest clinically than pathologically in that the patient suffered from violent præcordial pain radiating down the left arm. These attacks came on at night, and were as severe as those of angina pectoris. On one occasion, when free from pain, the man asked for a glass of water, and when the nurse returned to him with the glass, within sixty seconds, he had fallen over the edge of his bed dead, without having made any movement which could have placed an extra strain on the heart. The heart sounds were so obscure that a diagnosis of the lesion was impossible.

The next specimen was obtained from a colored man in middle life. Eight years ago he suffered from acute dilatation of the heart due to the lifting of a heavy piano. At that time he was seized with violent dyspnœa and nausea. This gradually passed off, and during the past eight years he has done his usual work. Recently, however, his heart muscle has failed, and he entered my wards suffering from præcordial pain and excessive dyspnœa, in which condition he died. The heart sounds during life were feeble and accompanied by a number of murmurs, which were loudest over the aortic and mitral areas. The area of cardiac dullness was very much enlarged. The heart is presented because of the peculiarity that there is a dilatation of all its cavities without any compensatory hypertrophy associated with the dilatation. The heart is therefore like the ox-heart of aortic regurgitation in size, but its walls are exceedingly thin.

May 26, 1898.

Spleen and Liver from a Case of Leukaemia.

H. A. HARE, M. D.

Notes of the Autopsy.—Body of an emaciated male; rigor mortis moderately marked. The abdomen was greatly distended; there was œdema of the feet, legs, penis and scrotum. On abdominal incision scarcely any subcutaneous fat was encountered; the muscles of the abdominal wall were very pale and friable.

The abdominal cavity contained 2000 c. c. of clear yellow fluid. Beneath old scars on the abdominal wall were numerous peritoneal adhesions; these scars were the result of frequent tapping. The peritoneum was somewhat thickened and reddened, and more opaque than normal. The surface of the omentum was roughened and scattered over its surface were numerous tumor-like enlargements which have a usual diameter of 1 m. m.

The lower margin of the liver in the right mammary line extended 5 c. m. below the costal margin; the upper margin extended to the upper margin of fourth rib; the left lobe of the liver extended downward 10 c. m. below the ensiform cartilage; the left margin of the left lobe extended to the left 5 c. m. beyond the left nipple line. The lower border of the spleen extended downward to within 7 c. m. of symphysis pubis; the right margin extended to the level of the nipple. The stomach lay in the angle formed by the separation of the inner borders of the liver and spleen, which was on a level with and just to the right of the umbilicus. This viscus was therefore pushed downward, forward and to the right of its normal position. The transverse colon was pushed downward, and occupied the position just anterior to the stomach; the left half of the transverse colon lay beneath the lower border of the spleen, and was consequently much prolapsed.

The right pleural cavity contained 100 c.c., of clear yellow fluid, but was otherwise normal, as was its fellow. The pericardium was normal and contained 20 c.c. of clear yellow fluid. The heart was large and very flabby; the left side was slightly contracted. The heart substance was pale and moderately friable. The right auricle was punctured and cultures made from the blood. The small amount of blood which exuded was fluid and light in color.

The spleen which, with the liver is here exhibited, is enormously enlarged. It weighed 5,500 grams. Its transverse circumference is 48 c.m., and its longitudinal 86 c.m. The organ is decidedly notched on its inner and outer borders. Over its surface are numerous white scars of irregular outline and size. On section its substance offers great resistance to the knife, it being much tougher than normal. As regards color the substance presents the usual peculiarities. In situations where scars occur it is found that this tissue extends down into the substance of the organ; these masses of scar tissue are irregularly pyramidal in form—the apices extending toward the central portion of the organ; they have the appearance of the remains of old infarcts. The splenic substance for perhaps a millimeter around these areas is much redder than in other situations.

The liver is enormously enlarged, weighing 6,300 grams. On section it is seen to be pale in color and of normal consistence.

The adrenals were somewhat enlarged and darker in color than usual, the substance of normal consistence.

The left kidney was displaced to the right, and lay in front of the spinal column, its long axis parallel to that of the spinal column. Over the surface of this kidney there were numerous cysts from .5 to 3 c.m. in diameter. The substance was pale and quite tough. The pryamids were not well marked off from the cortical portion, which was somewhat diminished in amount. The weight was 250 grams. The right kidney, with the exception that there were no cysts on the surface, was in the same condition as its fellow; weight 285 grams. The ureters were normal. The bladder contained a considerable quantity of opaque urine. Its walls were extremely thin but otherwise the viscus was normal.

The stomach and intestines were normal. Within the entire alimentary canal dark grumous masses were found which were evidently more or less altered blood. Within the mesentery several enlarged lymphatic glands were found; they were generally of a uniform faintly reddish color and contained but little fluid. In one instance a gland was found, the substance of which was rather grayish in color and scattered through its substance were numerous hemorrhagic areas.

A section of the right femur was made and the bone was found very hard. The marrow was much firmer than usual, and was of a reddish gray color. It seemed to be less firmly attached to the surrounding bone than usual. *May 26, 1898.*

A Case of Gall-Stone in the Common Duct, with Intermittent Fever.

FREDERICK A. PACKARD, M. D.

The patient from whom the specimen was removed entered Ward 5 in the Philadelphia Hospital on November 15, 1897, complaining of abdominal pain, jaundice, loss of appetite and constipation. His family and personal history presented no points of interest. He never had syphilis, and used alcohol and tobacco in moderation. On admission, he said that on the afternoon of November 10, he had a decided chill with dull pains in the abdomen. The chill continued for about twenty minutes, and was followed by a sense of warmth. During the attack he was nauseated, and vomited. The pain in the abdomen continued until after his admission; it was somewhat relieved by leaning forward or lying on the right side.

On admission it was found that the skin and conjunctivæ were decidedly icteroid. Save for a few moist râles and harsh breath-sounds posteriorly on the right side, the lungs were normal. The heart was normal, except for accentuation of the second aortic sound. The liver dullness began above at the sixth rib and extended downward to two fingers' breadth below the costal margin in the right nipple line. The spleen was not enlarged. The temperature on admission was normal, but rose to $99\frac{4}{5}$° in the evening. On the day after admission it rose from $101\frac{4}{5}$° at 8 a. m. to $103\frac{1}{5}$° at 11.30 a. m., without chill, and then slowly fell to normal by midnight. The urine was bile-stained, contained no sugar, a trace of albumin, hyaline, granular and epithelial casts. The jaundice and pain gradually disappeared, and he was discharged free from symptoms on November 29. This attack was looked upon as an ordinary catarrhal jaundice, and he was returned to the Almshouse Department.

He was again admitted, on this occasion to Dr. Salinger's ward, on February 16 of this year, because of irregularly recurring chills, followed by fever, jaundice and hepatic pain.

After his previous discharge to the out-wards, he had an undeterminable number of chills and a constant dull pain in the hepatic region not extending to the back or shoulder. On admission he was found to be slightly jaundiced. The hepatic dullness was found to be about normal in area, but there was some tenderness on deep palpation beneath the right costal margin. The spleen was not enlarged. In the first ten days after admission the jaundice lessened, the urine became of lighter color, the stools more dark. The temperature was normal until February 22, when it suddenly rose to 103$\frac{3}{5}$° upon the occurrence of a chill. It fell to normal before the next afternoon, to soon rise again to 102$\frac{3}{5}$°, with chill and a further rise of one degree. It then fell to normal and remained so until his discharge on April 11. During his stay in the wards on this occasion he had no severe pain, and from the notes it would appear that there was no increase of pain or of jaundice during or after the chills and rises of temperature. On this occasion the diagnosis of "impacted gall-stone" was recorded.

He remained in the out-wards, although jaundiced, until his admission to my ward on May 3. He was sent to the wards because of the occurrence of a chill with temperature of 104$\frac{2}{10}$° and pain in the right side in the hepatic region. He then said that he had been steadily jaundiced for three months.

On examination he was found to be a spare old man, giving his age as 73 years. The skin was slightly jaundiced, the conjunctivæ icteric. Arcus senilis marked. The pulse was rather collapsing, the radials were quite leathery. The spleen was not enlarged. The liver dullness in the right nipple line began at the upper border of the sixth rib and extended to a finger's breadth below the costal arch. Just below the tip of the ninth costal cartilage there was tenderness on pressure, with a decided feeling of fullness, but no distinct tumor. His blood was examined for the plasmodium malariæ, and the stools for gall-stones, but with negative results.

His temperature soon fell to normal and, with slight fluctuations, remained down until his death from asthenia on May 9. His urine contained bile and a faint trace of albumin, but neither sugar nor casts were found in either of two examinations. There was no perceptible increase of jaundice during his stay in hospital.

At the autopsy there were found healed areas of former trouble in the apices of the lungs, mitral sclerosis, intense atheroma of the aorta and chronic interstitial nephritis in addition to the following changes in the liver and bile passages. The great omentum was adherent to the peritoneum of the anterior abdominal wall by a firm old adhesion. The liver extended 7 c. m. below the xyphoid tip in the median line. There were a few old and quite strong adhesions binding the distended gall-bladder to the duodenum. The spleen was found to be small and soft. The gall-bladder was greatly distended. From it the cystic duct, distended to the size of the little finger, pursued a straight course to its union with the greatly dilated and distended hepatic duct. The common duct was distended to the size of the middle finger. No stone could be felt in the gall-bladder or ducts. On opening the duodenum there was noted a hard prominent swelling at the situation of the papilla of Vater. From the orifice of the papilla bile flowed slowly upon squeezing the gall-bladder. A probe passed into the orifice of the papilla struck a stone almost at the opening. This stone was found to be in the duodenal portion of the common duct. It is fully 1.5 c. m. in diameter and could be readily pushed upward into the lower portion of the hepatic but not into the cystic duct. The gall-bladder was filled with bile and measures 10 c. m. from fundus to the cystic duct, 4.5 to 5 c. m. in diameter. The hepatic duct, from the notch of the liver to the common duct, measures 7.5 c. m., its diameter being 17 m. m. The cystic duct is 5 c. m. long. 1 c.m. in diameter. The common duct is 8 c. m. long, 2 c. m. in diameter. Upon pushing the stone to the upper portion of the common duct and then squeezing the gall-bladder the calculus shot downward forcibly against the end of the duct, stopping the outflow of bile.

The liver weighed 1640 grammes. On the right upper surface of the right lobe are three deep furrows, evidently markings from the ribs. On the upper and posterior surface there were many friable adhesions. On the upper surface are three yellowish-white nodes measuring about 1 c. m. in diameter and slightly raised above the surrounding surface. On section these areas were found to be round areas of purulent infiltration about dilated bile ducts. Deep in the substance of the liver a few smaller and scattered areas of pus formation were found along the line of the ducts. The general liver tissue was firm but cut easily. The bile ducts are slightly too prominent, but the cut surface looks fairly normal. Smears of the pus about the small ducts in the yellowish-white areas described above show colon bacilli in great number and many streptococci.

The case is therefore an illustration of the occasional ball-valve action of a stone in the common duct to which attention has been so forcibly drawn by Osler and Fenger. It offers some points of especial interest that are worthy of note. The tremendous size of the gall-bladder, which during life gave the sensation of fullness without distinct tumor, is certainly unusual in cases of biliary calculus. The mechanism of the production of the dilated gall-bladder as well as ducts is readily seen in the specimen. From the cases described by Osler as illustrations of the ball-valve action of gall-stones this case differs, from the fact that the stone had a range of motion equal to the length of the common duct, instead of being limited to the ampulla of Vater. Its occasional impact against the orifice into the duodenum evidently produced complete or almost complete obstruction until the pressure behind it dilated the common duct sufficiently to allow of the stone readily being displaced upward. The fact that the cystic duct was too small to permit of the entrance of the stone into the gall-bladder is chiefly of interest because it prevented such a favorable termination as the accidental slipping of the stone into the larger and less important portion of the biliary apparatus, in which event comparative cure might have resulted from the stone being so placed as to be no longer capable of interfering with the free flow of bile. In spite of the presence of the stone, certainly during much of its time, in the duodenal portion of the duct there was no evidence of long-continued or intense pressure upon the outlet of the pancreatic duct. The latter was not markedly dilated, and there was no parenchymatous change in the gland, although a probe passed through the pancreatic duct impinged upon and scraped past the calculus as it was found at autopsy.

On opening the abdomen at the autopsy, the biliary passages were at once carefully examined. In spite of careful search it was concluded that no stone was present, and that the obstruction must have been produced by a healed duodenal ulcer, cancer of the head of the pancreas, or other extra-biliary cause. The calculus was only discovered upon slitting the duodenum. It is probable that the firm head of the pancreas prevents the ready appreciation of a stone situated at the extreme end of the duct in the duodenal portion. This failure to discover the stone upon leisurely examination in the cadaver, when its existence was clinically certain, shows how much more difficult such a search must be at operation during life, as in the first case alluded to by Dr. Osler, in his article in the *Lancet* of May 5, 1897, and related at greater detail in the *Annals of Surgery* for March, 1890.

The small areas of suppuration about some of the smaller bile ducts are evidently epiphenomena from secondary infection. That they played but little part in the clinical picture, is shown by the absence of all evidence of the constant intermittent or continuously elevated temperature of Charcot's hepatic intermittent fever. In this case the temperature was almost constantly normal or subnormal during his various sojournings in the wards, the dull monotony of his chart being relieved by occasional sudden rises and rapid falls of temperature. It is readily conceivable that, had the patient's vitality been greater, he might have presented in time the more complicated picture of Charcot's hepatic intermittent fever (pyæmic) with intercurrent more abrupt rises from sudden obstruction of the outlet of the common duct (toxic or, possibly, simply irritative).

It is interesting to observe the uniform and great dilatation of the common duct which doubtless caused his attacks of complete obstruction to be of short duration, because of the free mobility of the stone upon increase in the size of the duct, probably following shortly after each attack of obstruction.

May 12, 1898.

A Case of Acute Appendicitis with Secondary Pylephlebitis and Multiple Abscesses of the Liver.

J. C. GITTINGS, M. D.

The following case occurred at the German Hospital, Philadelphia, in the service of Dr. John B. Deaver, who has kindly permitted me to report it.

J. H. K., *aet.* 33, a railroad engineer, was admitted to the surgical ward of the German Hospital on January 13, 1897, at 9 a. m., having made the trip from Lewistown, Pa., a distance of one hundred and fifty miles, during the preceding night. The patient had suffered a probable attack of acute appendicitis six months previous. Following free purgation complete subjective recovery resulted in a few days. He gave a history of internal hemorrhoids several years ago; they had however been quiescent for at least three years.

Three days before admission he experienced another acute attack of abdominal pain, which, at the end of twenty-four hours, became localized in the right iliac fossa, and was associated, from the start, with repeated attacks of vomiting and obstinate constipation. On the second day of the attack the pain became less paroxysmal but more severe, and he became markedly prostrated. He was sent to the hospital for operation, and while on the train, without obvious cause, he had a severe chill, followed by a slight sweat and increased prostration.

On admission his temperature was $100\frac{1}{5}$°, his pulse 88, full and strong. The abdomen was not distended, but there was marked rigidity of the right rectus muscle, with excessive tenderness at McBurney's point. No mass could be made out, but the above conditions prevented any but the lightest palpation. Immediate operation was performed by Dr. Deaver. The appendix was found, pointing southwest and completely surrounded by a mass of omentum, to which it was partially adherent. Separation was carefully effected, and the appendix found to be soft, distended and gangrenous for the distal two-thirds of its length. It was removed, without rupture, together with the mass of omentum and inflammatory exudate. Twenty minutes after the patient's removal to bed, instead of a condition of shock, the temperature was found to be at 102°, the pulse 100, strong and full. Shortly afterward he had a pronounced chill, followed by a slight sweat.

On the following day he complained of the usual pain following an appendisectomy. His thirst seemed rather excessive. The temperature again rose to $102\frac{2}{5}$°, the pulse was 96, there was no chill. The stomach was retentive, and the abdomen was not distended, flatus passing easily. The temperature ranged high all the following day, and at noon he had a slight epistaxis (no history of such occurring during health). The urine was normal. The next day temperature ranged above 103° all day. The spleen appeared slightly enlarged on percussion, but could not be felt. At 5 p. m. he had another chill, followed by a drenching sweat, with a temporary fall of temperature. Another chill and sweat occurred on the following morning, quickly followed by high fever. He complained of slight pain and tenderness on pressure over the region of the gall-bladder. No additional enlargement of the spleen could be detected. The wound was dressed and found to be in good condition. A blood examination for the plasmodium of malaria was negative. The red cells were 4,125,000, white cells, 50,000 per c. m. m., the hemoglobin 85 per cent.

The urine then contained traces of albumen, red blood cells and hyaline casts. On the next day a dusky tint appeared on the skin and conjunctivae which by evening had developed into a distinct jaundice; the temperature at this time reached 104°. A repetition of the nose-bleed occurred and also another chill. Upon the occurrence of jaundice, together with the localized tenderness, and general septic condition, a diagnosis of pylephlebitis suppurativa was made. The nose-bleed recurred upon several successive days and an intestinal hemorrhage occurred once, while several of the later stools presented a tarry appearance.

The patient's condition became rapidly worse during the next week. The cardiac sounds became feeble, the pulse became rapid and running, there was a constantly leaky skin. The liver dullness was increased, but at no time became markedly so. The jaundice persisted. The total quantity of urine was about 300 c.c. per diem, and contained bile-stained epithelium and epithelial casts. The abdomen became tightly distended and tympanitic throughout. The patient became markedly septic, then comatosed, and died on the nineteenth day of the disease.

Autopsy. The body was well nourished but deeply jaundiced. The abdominal wound was firmly healed. There was a cloudy change of the myocardium. The lungs were congested, the right was firmly adherent at the base.

The omentum was adherent to the cicatrix in the cæcum, and to the mesentery, inclosing a small pocket of pus. No opening existed in the cæcum, however, the cicatrix being impermeable. The stomach and remaining intestines presented no abnormality except congestion of their mucous membrane, this being more marked in the latter. The spleen was large, congested and pulpy (septic); no infarcts existed. The kidneys were congested and showed parenchymatous changes. Both ureters were dilated just before reaching the pelvis.

The liver exhibited a perihepatitis, with adhesions upon its upper surface. It was enlarged, and soft, presenting a mottled appearance from the existence of numerous small abscesses under the capsule. On section, the portal vein, from its very origin down to its minute ramifications, appeared filled with pus. The intervening hepatic structure was intensely congested, presenting here and there small necrosed areas and purulent collections, communicating with the veins. The gall ducts were patulous. Cultures from this pus revealed the presence of staphylococci and streptococci.

Sections of the appendix showed the presence of an old stricture near the base, causing complete occlusion, beyond which dilatation existed. The dilated portion showed thinning and gangrene of the walls, with abrasion and in some places entire absence of the mucous membrane. It was filled with detritus,—epithelium, inspissated mucous, etc.

The case is an unusual one, although suppurative and gangrenous appendicitis is quoted by all recent authors as being the most frequent cause of suppurative pylephlebitis, and presents several noteworthy points of interest. The rapidity with which necrosis occurred, quickly followed by portal infection, is unusual. The average time for the latter is variously placed from a few days to one or two weeks. In the present case it was less than three days, probably shortly before the occurrence of the chill while the patient was being brought to the city. Further proof of its pre-operative origin can be found in the high temperature immediately following the operation, in place of the usual condition of shock. The cause of this rapid infection probably corresponded to the cause of the rapid necrosis of the appendix, namely, highly infectious material. The condition of the appendix prior to the second attack was one of passive dilatation, behind the stricture, from the mass of debris, causing thinning and even partial destruction of the walls. Infection occurring, and the appendix being unable to empty itself on account of the stricture, rapid necrosis was the natural consequence. The pylephlebitis was probably the result of embolism from the veins of the appendix, which emboli were distributed to all of the large branches of the portal vein. The rapidity of the infection, together with the absence of inflammatory change in the extra-hepatic portion of the vein, would lead to this assumption. Had thrombosis occurred, the symptoms of portal obstruction would have been more marked, together with proof at autopsy. The absence of severe pain is explained by the fact that the peritoneal covering was not early involved. The perihepatitis found at the autopsy doubtless occurred during the latter days of the disease, when the mind was clouded. The septicæmia was marked and resulted in death without the occurrence of secondary embolism, in contradistinction to primary hepatic phlebitis, where pulmonary embolism is almost a constant sequence. The absence of portal obstruction is of importance. The hemorrhages were apparently largely septic, occurring as they did, almost as often from the nose as from the intestine. Vomiting, ascites or diarrhœa were absent. The jaundice was due to involvement of the biliary radicles, and was of the intrahepatic form. That the stools contained coloring matter was probably due to the disintegration of blood.

DISCUSSION.

DR. PACKARD: I have seen two cases of hepatic abscess in connection with appendicitis. The first case presented chills, irregular fever and jaundice, without signs of appendicitis. The autopsy revealed a collection of pus ascending the lumbar muscles, portal thrombosis and multiple small hepatic abscesses. In the second case, which presented definite signs of appendicitis, there was complete thrombosis of the portal vein, together with a large abscess in the liver, following peforation of the appendix by a pin. In neither case was there any ascites.

DR. DOUGHERTY: I have seen four cases of hepatic abscess in connection with appendicitis, all of which occurred in subjects with simple catarrhal lesions. This complication can certainly occur in the mildest cases as well as in the gangrenous, but I have never seen it follow purulent conditions of the appendix. Kolisko, in Vienna, told me that he had never seen abscess of the liver occur in the course of an acute suppurative appendicitis.

DR. GITTINGS: Most of the cases reported in the literature have been noted in connection with gangrenous appendicitis.

DR. SAILER referred to a case of typhoid fever in which he had obtained a pure culture of the bacillus pyocyaneus from one of the mesenteric lymph glands. There were no evidences of suppuration.

May 12, 1898.

A Case of Intracanalicular Fibroma of Both Mammary Glands.

F. SAVARY PEARCE, M. D.

1. In Volume xvii, pp. 125 and 157 of the "Transactions of the Philadelphia Pathological Society," I made two macroscopic and microscopic reports upon a case of intracanalicular fibroma of the mammary gland, with no recurrence up to two and a half years after amputation of the breast. The patient, a woman *aet.* 53 years, married and a multipara, was extremely emaciated and cachectic. The tumor had been present for over thirty years, and at the time I saw and removed it (in 1895) had many appearances of a morbid neoplasm. The later clinical history of this case is interesting. The woman regained excellent health after the operation, her cachexia left her entirely and she remained well several years. Then vague abdominal signs and symptoms developed (disturbances of digestion and fatty stools). Persistent nausea, vomiting and epigastric pain finally ensued; and upon palpation tenderness and a sense of resistence was made out in the right upper quadrant of the abdomen.

Nothing could be positively determined, however, by physical diagnosis or semiology. After death a thorough search revealed only an enlarged and fibroid condition of the pancreas. The uterus, while not enlarged, was what might be properly called sclerosed. I regret there has not been made any microscopic sections of the pancreas to know exactly the histologic nature of the fibrosis. The site of the breast wound was healthy, nor were the lymphatics anywhere enlarged.

2. The second case, detailed to-night, is that of an American, nurse, single, *aet.* 28 years. In 1892, she consulted Dr. DeForest Willard on account of a lump the size of the end of the thumb in the left breast above and to the outside of the nipple, then painful and discovered accidentally. Dr. Willard advised no treatment. Being a philosophic woman she thought no more of the growth, although at times it produced an unpleasant drawing sensation. She went into difficult surgical nursing in 1894. In 1897 an increase in size of the tumor became apparent, but it did not begin to grow measurably until the past year, following a severe attack of grippe.

Since December, 1897, the tumors have very manifestly increased in size, and recently have become quite painful and tender, and always remarkably more tender just before the menstrual periods. She again lost flesh and strength. The larger tumor in the left breast became the size of a goose egg, non-ulcerated and was exquisitely tender if any forceful pressure were made. There were two smaller tumors in this breast. The nipple was not retracted. No lymphatic glands were enlarged. A small nodule of similar nature appeared also in the right breast occupying an acinus above the nipple, and a second one below, the size of the end of a thumb, also painful prior to the menstrual epochs. The patient was all the time conscious of the right breast, and that shooting pains in both breasts exist at intervals.

Dr. William H. Parish with Drs. Willard and H. R. Wharton agreed as to the adenomatous nature of the tumors and the advisability of removal. From the previous experience I made the diagnosis of an intracanalicular fibroma.

On April 15, at the Presbyterian Hospital, Dr. Parish removed the entire left gland, and then enucleated the tumors above and below the right nipple. The surgical recovery has been complete, quick and uneventful. The patient was discharged in three weeks and is now in recovering health.

Dr. J. Dutton Steele has kindly made a series of sections of the growths which are here shown, determining them to be intracanalicular fibromata. There is no history of any tumor in the family of this patient, but one sister died of rapid phthisis.

3. The third case occurred in a Hebrew, multipara, *aet.* 51, who reported November 24, 1897, in a myxœdemoid state and suffering from incipient rheumatoid arthritis. She gave a history of "tumors of both breasts in her mother." About five years ago pain in the breasts before the menstrual periods attracted her attention; tumors were found in them, and in June, 1894, one breast, and that fall the other breast, were removed. There has been no recurrence. The climacteric was established in 1894, but she continued to bleed from the womb, and Dr. E. E. Montgomery removed the uterus and ovaries in 1895. Since, after three years, there has been no recurrence anywhere, without knowledge of a microscopic study, I am inclined to think the tumors fibroid and hereditary. Thyroid extract has reduced the myxoedema in this woman.

4. Dr. Parish has told me of operating upon a Cuban woman (single, *aet* 20 years), almost one year ago, enucleating breast tumors of a similar nature to those described in the second case, and that now a second operation will have to be resorted to for total extirpation of the breasts, since the growth has progressed in the other accini. In this case Professor Guiteras pronounced the growths fibro-adenomata. Ten years ago this patient's mother had a tumor removed from the breast and is perfectly well to-day.

Malignant growths of the mammary glands are far the more common, but the cases cited here point to benign tumors being more frequent than supposed, and in the light of the clinical history shows the paramount importance of careful microscopic study before a positive diagnosis or prognosis can be given.

CONCLUSIONS.

Heredity is evident in two of the cases here reported. The diatheses of gout and phthisis occurred in two of the family histories. Three cases were under thirty years of age and one was over thirty years of age when the tumors began to develop. The slowness of growth in all and absence of metastasis were the points in favor of non-malignancy in the four cases. In one the pancreas and uterus were also involved in an apparently similar process. In two cases lowering of general health seemed to be the occasion for more rapid growth. The pain experienced in these two cases at the menstrual periods shows the close sympathetic nerve relation between the genitals, breasts and general vaso-motor nervous system.

It is evident that an increasing exactness of pathology will better classify benign tumors in the future. Perhaps some of the so-called sero cystic tumors of Sir Benjamin Brodie are but forms of adeno fibroma. As to heredity in fibroid conditions and other tumors, the theory of Conheim seems more and more to have a substantial basis.

DISCUSSION.

DR. STEELE.—These neoplasms are in strict pathology adenomata. The process represents an over activity of the epithelium of the proliferating glands and consists of buddings of papillomatous growths into the lumen of the acinus. The amount of connective tissue varies with the stage of the process. The youngest growths present a distinctly dendritic appearance. As the papilloma enlarges so as to completely fill the lumen it looses its branching form from pressure and the epithelium of the growth is pressed so firmly against that of the wall of the acinus that it is often hard to recognize that there are two layers present, hence the misnomer intracanicular fibroma. The sections show all the stages of the process. I have recently seen five examples of such neoplasms, four in the mamma and one in the endometrium.

May 12, 1898.

Rupture of the Aorta, with Flap-like Dissection.

J. D. STEELE, M. D.

The specimen was obtained from a well-preserved man of about 45 years of age. Nothing is known of his previous hi tory except that the cause of death was said to be acute arsenic poisoning of 24 hours duration.

The pericardium is normal. The heart is somewhat dilated and slightly hypertrophied. The left ventricle is 12 m. m. in thickness and the right 4 m. m. Both auriculo-ventricular orifices are dilated, and each admit four fingers. The myocardium is apparently in an advanced stage of fatty degeneration. There are marked striations upon the papillary muscles. There is a considerable amount of interstitial myocarditis. There are a few spots of atheroma upon the anterior leaflet of the mitral valve and upon the bases of the aortic leaflets, but the valves appear competent. About 3 c. m. above the attachment of the aortic valves, on the posterior wall of the aorta, a piece of the lining of the vessel has been dissected up in a horseshoe shaped flap, which is 2 c. m. wide at its attachment and 2 c m. long. The edges of the flap are slightly ragged. The flap itself is perhaps 1 m. m. thick on an average, but varies considerably, and when held to the light is seen to be much denser in some places than in others. These areas seem to be fibroid thickenings of the intima that have been dissected up. The portion of the aortic wall that has been laid bare by the raising of the flap is smooth and opaque, and of a light yellow color, and is evidently the seat of an advanced fatty degeneration. The whole aorta is very much thickened, and its intima everywhere is studded by plates of fibrous tissue, which are yellow and opaque. The whole interior of the vessel is a typical picture of arterial sclerosis. One centimeter below the lower end of the flap is a small oblong atheromatous ulcer.

Microscopic examination of a piece of the aorta taken from the edge of the eroded area shows that the rupture involved about half the intima. This layer is very much degenerated, and the nuclei of its connective tissue almost invariably refuse to stain. The endothelium is entirely destroyed. The intima is thickened and there is a scanty number of round cells scattered through it. Indeed, these are the only cells to stain uniformly. The media and adventitia are thickened and show excess of connective tissue. There are collections of round cells in several areas in the outer layer, always around the blood-vessels of the arterial coat.

The pleural sacs were obliterated on both sides. The lungs were much congested and œdematous. The kidneys were cyanosed and contracted. The liver was fatty and cyanotic. The gastric mucous membrane was smoothed and glazed and appeard thin, and was probably in a condition of chronic atrophic gastritis. There was no sign of acute inflammation, nor did it suggest the presence ot any acute corrosive poison. The duodenum and intestine, as far as examined, did not present anything unusual. It would appear, in view of the indefinite history, that the cause of death must be sought in the degeneration of the heart muscle and not in the arsenic, if any such material were taken.

The body was in a good state of preservation, rigor mortis was still present, and the autopsy was performed in cool weather and thirty-six hours after death. The aorta was not examined or touched until it was laid open, and the peculiar condition found in it could not have been produced by any post-mortem handling.

The degenerated condition of the aorta that was found microscopically would appear to be a sufficient explanation for the spontaneous rupture of the aorta and the starting of the dissection. If the rupture had extended deeper so as to embrace more of the tissue of the vessel wall and so allow of the raising of a stronger flap, it is probable that a dissecting aneurysm would have been formed, but the flap tore so easily that it gave away before the force of the blood current, with the result that is here seen.

It is possible that the condition is a stage in the formation of a so-called atheromatous ulcer, in which the fatty material is being thrown off in a large piece instead of as granular debris. The edges of the smaller ulcer below appear exactly similar to those of the larger erosion. The leading authorities upon the pathology of the arterial system do not refer to this condition, though Ziegler in his 1895 edition has a plate of partial rupture of the aorta that resembles it, except that there is no flap. Both Orth and Ziegler state that partial rupture of the aortic wall is not uncommon. In view of the fact that there was no evidence of acute poisoning in the gastro-intestinal tract, it does not seem possible that a large amount of arsenic could have been taken, and that it could have had anything to do with the extensive fatty degeneration of the cardio-vascular system.

DISCUSSION.

DR. PACKARD referred briefly to a case, the specimens of which will later be presented to the Society, of an old man who was suddenly attacked by a condition of collapse with severe epigastric pain. There was the murmer of aortic stenosis, and a provisional diagnosis of mesenteric infarction was made. At the autopsy the posterior mediastinum was found filled with blood which had proceeded from a transverse rupture of the aorta. At the site of the rupture the aorta was not atheromatous, but the intima was curled up into the lumen of the vessel.

May 12, 1898.

Obliteration of the Orifice of the Right Coronary Artery.

J. G. TAYLOR, M. D.

E. G., *æt.* 25, female, was a patient in the Pennsylvania Hospital under the service of Dr. Meigs. Although she had had, two and a half months before admission, a miscarriage, no history of syphilis could be obtained. Her illness dated from six days before admission, when she suddenly had very severe pains about the præcordia, radiating down the left arm, "which were so severe that she fainted" (husband's statement). Since that time she has had continual headache and much dyspnœa.

On the morning of admission she had been suddenly seized with very severe præcordial pain, and was brought into the hospital in a state of partial collapse, from which she seemed to revive. When I began to examine her she complained of some pain, answered a few questions, and suddenly had convulsive twitchings of the muscles of the face, these quickly became general, and death ensued in a few moments.

At the autopsy the following conditions were noted: The lungs, liver, spleen, kidneys and intestinal tract were normal. The heart was normal in size, the pericardium contained the normal amount of fluid. The mitral valves were healthy. In the aorta above the valves for one and a half (1 ½) inches was a large deposit of calcareous plates. The orifice of the right coronary artery was entirely obliterated. The artery was found upon dissecting down behind the valves, but it was small, appearing as if it were a small branch. The opening of the left coronary artery was very small, about one quarter (¼) the natural size, but the artery was found to be normal in size and could be readily traced with a probe, admitting a large size probe. The aortic valves were thickened and calcareous.

DISCUSSION.

DR. MEIGS: The case is interesting for two reasons: First, as another instance of the connection of angina pectoris with disease of the coronary arteries, and second, on account of the occurrence of angina in so young a person. It is curious that the left coronary artery, which is usually much smaller than the right, should have been able to carry on the circulation. The opening of the coronary artery from the aorta was, owing to the atheroma and calcareous deposits in the aorta, of not more than one-third the natural size. The aorta, with the exception of its commencement, was entirely free from disease. It is strange that atheroma and calcareous deposits should have been of such an advanced degree and yet confined to so small an area.

May 12, 1898.

Multiple Abscesses of Lung following Pneumonia and Empyema: Ulcerative Endocarditis.

J. G. TAYLOR, M. D.

S. T., male, *æt.* 20. Admitted to Pennsylvania Hospital in the service of Dr. Meigs.

The present illness dated five days previous to admission and was of sudden onset, with chill, cough, blood-streaked expectoration, dyspnœa and fever.

Pneumonic consolidation was present from the apex to the base of the left and in the base of the right lung. The urine contained albumen, hyaline and granular casts. The temperature was 104°, the pulse 100, the respirations 60 to the minute. There was improvement until the tenth day after admission, when the temperature rose suddenly, and marked hectic symptoms intervened. Anteriorly, in the præcordial region there was marked bulging of the chest, and on the seventeenth day after admission he was operated upon by Dr. Ashhurst, who made an incision in the third interspace in the nipple line, and evacuated a large amount of pus, a drainage-tube being inserted. Two days before death a very profuse hemorrhage occurred through the tube. He died four weeks from date of admission. A few days before death, examination by Dr. Meigs revealed signs of pneumonic consolidation over the whole of the left lung, and posteriorly over the lower lobe of the right lung.

On autopsy the pleura and lung were found tightly adherent about the opening. The pleura upon the whole of the left side was also firmly adherent. The drainage-tube had forced quite a large space in the upper portion of the left lung, the cavity being much larger than the tube, hence the lung must have moved to and fro upon the tube during the respiratory movements. At the point of entrance of the tube and above, to the apex of the lung, there was much plastic lymph, which had been a locular empyema. Near the apex of the lung was an abscess in its substance, which had discharged downward through the tube. Throughout the lung there were several abscess cavities near the surface, and none of great size. Both lungs were almost everywhere in a state of soft hepatization.

The right pleural sac contained about two quarts of clear serum. The right lung everywhere was free from adhesions.

All the abdominal organs were displaced downwards, the greater curvature of the stomach being upon a level with the umbilicus. They presented no noteworthy changes.

The pericardium was roughened upon the external surface. There were no adhesions. The sac contained four ounces of clear straw-colored fluid. Upon all three cusps of the aortic valves were minute fresh vegetations. There was a button-hole mitral valve, the opening being so small as to admit only one finger. Behind the posterior leaflet there was a large calcareous deposit. The pulmonary artery appeared larger and thicker than the aorta, which looked unusually small. The right auricle was greatly distended with a clot. The right ventricle seemed relatively more enlarged than the left, and its walls relatively more thickened.

May 12, 1898.

Cancer of the Caecum.

A. C. MORGAN, M. D.

This case occurred during the present service of Dr. F. A. Packard, at the Philadelphia Hospital.

The patient, C. M., female, Irish, 60 years, was admitted May 2d, 1898.

The family and previous personal history were negative.

The chief complaint upon admission was diarrhœa; there was a swelling in the right iliac region. She had had more or less vomiting of undigested food since last October, vomiting without relation to time of eating or character of food, and had steadily lost flesh since that time. At Christmas she noticed a mass in the right side of the abdomen. Soon after this, she lost a great deal of blood from the rectum, suffering no pain whatever during this occurrence. The swelling of the abdomen then decreased somewhat, and she felt quite well for some time. She has had a persistent diarrhœa for five or six weeks, at times with incontinence. Considerable pain accompanied the movements, which were yellow in color, and contained no blood. Prior to this time her bowels had been very constipated. One week before admission her legs had begun to swell, the right leg being larger than the left. She was emaciated to a marked degree. The pulse was small, and occasionally dropped a beat; the vessels were markedly atheromatous.

The abdomen was collapsed and wrinkled; soft and tympanitic everywhere, except over an area in the right iliac region, about the size of an egg, which was dull. There was there a mass which apparently was connected with the caecum, and which was hard, nodular and movable. The heart was irregular in action, but presented no murmurs.

At 3 p. m., on the day of admission, the patient was seen by Dr. Packard. At that time the abdomen was soft and relaxed. At 5.40 p. m., she complained of pain in the abdomen. At 7 p. m., the abdomen was much distended, tympanitic, and painful on pressure. Her temperature was 101° F. A surgeon at 8.30 p. m, diagnosed a temporary obstruction. The following day the bowels moved and the stools contained a small amount of blood, mucus, and undigested food, and some shreds which looked like tissue. A vaginal examination showed the uterus and adnexa to be normal, and not connected with the tumor mass.

The notes made by Dr. Packard are as follows: The most prominent portion of the tumor lies in a line drawn midway between anterior superior spine and the umbilicus. Apparently the apex presents forward, as a border mass can be felt deep down in the abdomen. Pressure in the right loin fails to move the mass. The mass is slightly movable, very firm and is distinctly separated from the hepatic dullness by a loudly tympanitic zone. The stomach seems very much dilated, but by auscultation and percussion it seems not to be connected with the tumor. The kidneys cannot be mapped out. Vomiting occurred daily, until death, two weeks later.

The autopsy was performed by Dr. Packard three hours after death. The body was poorly nourished. The abdomen was slightly prominent.

Both pleural cavities were attached by old adhesions. The heart was normal save for senile changes. The bronchial glands were enlarged and pigmented. The *right lung* was very large, firm, and solid, particularly in middle lobe, a portion of which sank in water; the rest of the lung was very œdematous. The *kidneys* were large and pale, the pyramids were intensely injected, contrasting strongly with the pale cortices. The lower edge of the right kidney was adherent to the caecal tumor, the capsule tearing at this portion. The *stomach* was somewhat dilated. The dilated gall-bladder projected 12 c. m. below the costal margin. At its fundus there was a firm adhesion to the parietal peritoneum.

The caecal tumor was found to be densely adherent to the false pelvis and to the psoas muscle. Attached to the median surface of the caecum was a loop of small intestine.

On removing abdominal contents entire, it was found that the mass was adherent to the psoas muscle, from which it could not be separated without rupture. The mass also surrounded the aorta near the cœliac axis. The *duodenum* communicated by a large opening, with firm fibrous boundaries, with the cavity of head of caecum.

Lying just within this opening, in the cavity of caecum, were two prune stones.

On completely opening the duodenum, it was found that 11 c. m. below the pylorus, where the duct takes a course to the left, was an opening 3 c. m. in diameter, leading directly into the cavity of the caecum. The edges were decidedly infiltrated, and there was involvement of the wall of duodenum, so occluding its calibre as just to admit the little finger. The orifice of the bile duct was free from involvement and opened directly above the orifice into the caecum.

The whole caecum, excepting a small portion of the anterior surface, was involved in a dense, hard, new growth. The ileo-caecal orifice was surrounded by the infiltration, so that its calibre was very much reduced. On opening the caecum it was found that the greater part of the wall was occupied by ragged, sloughing tumor, covered with a black material. There was one band of fairly normal mucous membrane remaining, measuring about one and a half inches across.

The ascending colon was not involved, but the caliber from the caecum to the ascending colon was less direct, and of smaller size than is that into the descending colon. On the posterior surface there was a large, ragged opening, where the caecum was torn away from the adherent psoas muscle during its removal, with shreds of gangrenous muscle fibre still attached to it.

The *pancreas* lay in a mass of hard enlarged glands, but the organ itself was free from involvement. Surrounding the aorta, and the ascending cava, in the region of the cœliac axis there was a group of very hard glands. No infiltration of the vessel walls was evident.

The *gall bladder* was distended, and contained 100 c. c. of bile and also forty-five gall-stones of alternating brown and white color, of uniform character, faceted, and varying in size up to 12 m.m. in diameter. They were not encapsulated, and there was no impaction. The cystic, hepatic, and common ducts were all greatly dilated and patulous. Just at the beginning of the duodenal portion of the common duct was a mass of hard, lymphatic glands capable of obstructing the free flow of bile, but evidently not producing permanent and complete occlusion. The lining membrane of gall-bladder was congested and a little roughened.

The general color of the liver was pale. On its upper surface was a deep notch, extending 3 c. m. inward (a malformation or rib-mark). On the upper surface of right lobe there was an area of peri-hepatitis, beneath which, on section, were found nodules of two kinds:—one, almost circular, measuring 5 c. m. in diameter, of a marble red color, having in its center a branch of the vena cava; the other form of growth was just beneath the surtace, and of yellowish white color, without manifest connection with the vessel, composed of two portions, each measuring approximately 1 c. m. in diameter. One of these was almost perfectly oval; the others of irregular shape, with three rounded branches. The general surface of the section was pale, slightly "nutmeg," and evidently very fatty.

DISCUSSION.

DR. EDSALL recalled a case of secondary carcinoma of the liver in which ante mortem an apparently independent tumor was to be felt in the right iliiac fossa, separated from the enlarged and irregular liver by an area of resonance. There were no gastric signs or symptoms, therefore the illiac tumor was held to be the primary growth, situated in the intestine. On autopsy the primary growth was found to be gastric, while the mass in the right illiac fossa was a lower portion of the enlarged liver constricted off from the main organ, a loop of intestine lying over the constriction and thus accounting for the physical signs.

Dr. Steele described an example of short-circuiting of the intestine, in which the duodenum and the ilium were adherent to an enlarged lymplatic gland which broke down and ruptured into each, with the persistance of the channel.

May 26, 1898.

A Case of Cancer of the Rectum and Liver.

WM. PEPPER, JR., M. D.

The specimens are from a woman of 39 years of age, who was admitted to the Philadelphia Hospital on April 30, 1898, complaining of pain in and swelling of her abdomen and of vomiting. She had had no subjective symptoms until seven weeks prior to admission, when her appetite began to fail, and her abdomen to distend; in the course of a week vomiting set in, followed by a severe attack of jaundice, lasting two weeks. She lost weight during the seven weeks, and also became quite anæmic. After her admission to the hospital she suffered greatly from vomiting and agonizing pain in the abdomen. She had no symptoms of rectal involvement; there was no pain in that region nor blood in the stools, there was no constipation. The liver could be plainly felt, extending down to the umbilicus; it was hard, and a few scattered nodules could be felt on the surface. Her stomach contents were twice examined with negative results. The blood count was 1,620,000 red cells, 10,000 whites per c. m. m., and 50 per cent of hæmaglobin. She died May 21.

At the autopsy the body was considerably jaundiced, especially the face, trunk and eyes. Rigor mortis was slight. There was a bed sore upon the right hip, and some œdema of the legs. The abdominal cavity contained about three pints of a turbid reddish-brown fluid. The liver extended 7½ c. m. below the costal margin in the right nipple line; in the median line 9 c. m.; in the left nipple line 6 c. m. There were some solid masses in the omentum, of a greenish color. There were adhesions between the mesocolon and the abdominal wall, and between the liver and the diaphragm. The epiploic appendages had projecting from their surfaces small tumors which were pendulous and soft. The uterus was displaced to the left and was adherent to the rectum. The left pleural cavity contained a half-pint of bloody fluid. The lung on that side was retracted and partly collapsed, but there were no adhesions. The right lung was adherent above and very much compressed by the liver. There was a small amount of bloody fluid in the right pleural cavity. The heart was small, weight 300 grammes. The intima of the aorta showed slight sclerosis. There were three openings for the coronary arteries. The mitral valve exhibited a few patches of sclerosis. The left ventricle was dilated, the muscular substance soft, flabby and pale, its wall measured 10 m. m. in thickness; the right 3 m. m. The left lung presented three or four cancerous nodules in the lower lobe, while the right lung contained one nodule the size of a large pea, of a grayish white periphery and a yellow butter-like center. The spleen was not enlarged, and contained three cancerous nodules, which, on section, presented a peculiar gelatinous colloid appearance; the nodules were well circumscribed, although not encapsulated. The kidneys were slightly sclerotic and contained small cysts; the right exhibited several, the left one apparently cancerous nodule.

The common bile duct is patulous. Just at the mouth of the duct, and just to the right of the opening, is a yellowish nodule about the size of a large pea. This nodule is closely connected with the mouth of the duct. About one inch from the duodenal papilla there is a circular opening somewhat concealed by an overhanging valvula conivens, measuring 1 c. m. in diameter. Its edge is rounded, but not elevated or indurated. This ulcer extends down to the serous coat. The common duct lies between the under surface of the liver and a cancerous nodule, probably a lymphatic gland, which must have produced pressure during life. The liver is greatly enlarged, weighing 4650 grammes, and is filled with large secondary growths, some as large as an apple; most of them are quite firm, and there is but slight umbilication. More nodules are upon the lower surface than upon the upper. Both lobes are equally involved. The pancreas was small, but showed no cancerous nodules. One or two glands of the mesentery are the seat of cancerous growths. The rectum, which with the liver is here exhibited, is the seat of a large ulcerating soft cancer encircling the entire lumen. It begins about 8½ c. m. above the anus, measures 9 c. m. in height, and at its widest part measures 9 c. m. in width. The surface is deeply ulcerated, greenish in color, excavated deepest in the median part of growth. The lower edge is everted and hangs somewhat down towards the anus. In the upper part of the growth there are some polypoid projections. The wall of the rectum is greatly thickened, measuring 4 m. m., and is very soft and friable. The uterus and bladder were not involved.

May 26, 1898.

Obstruction of the Cystic Duct.

WM. PEPPER, Jr., M. D.

The specimen was taken from a subject who died of phthisis, with marked pulmonary lesions. There was no jaundice.

The gall bladder is greatly distended; it extends 5 c. m. beyond the edge of the liver. It is pear-shaped, and its long diameter is 9 c. m. Just at the proximal side of the junction of the hepatic duct with the cystic duct, the latter is occluded by a gall stone, the size of a large pea. It is impossible to force any bile from the gall bladder into the duodenum. The fœcal matter is intensely bile stained. The gall bladder contains a large number of gall stones.

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TABLE OF CONTENTS.

RIESMAN, Congenital Hydronephrosis.—COPLIN, Primary Cancer of the Liver.—COPLIN, Diffuse Tuberculosis of the Testicle.—SAILER, A Contribution to the Pathology of the Choroid Plexus.—SPILLER, A Tumor Resembling in Structure the Choroid Plexus.—SPILLER, On Amyloid, Colloid, Hyaloid and Granular Bodies in the Central Nervous System.—SPILLER, Sections from the Sternocleidomastoid Muscle in a Case of Congenital Torticollis.—SAILER, Alterations in the Spinal Cord in Azoturia.—PACKARD, A Case of Rupture of the Aorta.—ESHNER, Enlarged Cirrhotic Liver; Dilatation of the Heart, Atheroma of the Aorta, Pulmonary Infarction.—PACKARD, Pseudo-Parasites in the Stools of a Child.—ESHNER, Chronic Interstitial Nephritis (Cystic Kidneys): Hypertrophy of the Heart, Hemorrhage into the Pons Varolii.—JAMISON, Cancer of the Head of the Pancreas.—RIESMAN, Intestinal Obstruction by a Peritonitic Band.—STEELE AND GRIFFITH, Cystic Kidneys.—SAILER, Calculus of the Spleen; Typhoid Ulcer in the Appendix.—PACKARD, Minute Hemorrhage into the Pons; Multiple Abscesses of the Liver Secondary to Chronic Purulent Peritonitis.—ARNOLD, Colloid Cancer of the Stomach; Atrophy of the Kidney from Obstruction of the Ureter.—WILLIAMS, A Carcinomatous Polyp of the Cervix Uteri.

Congenital Hydronephrosis.

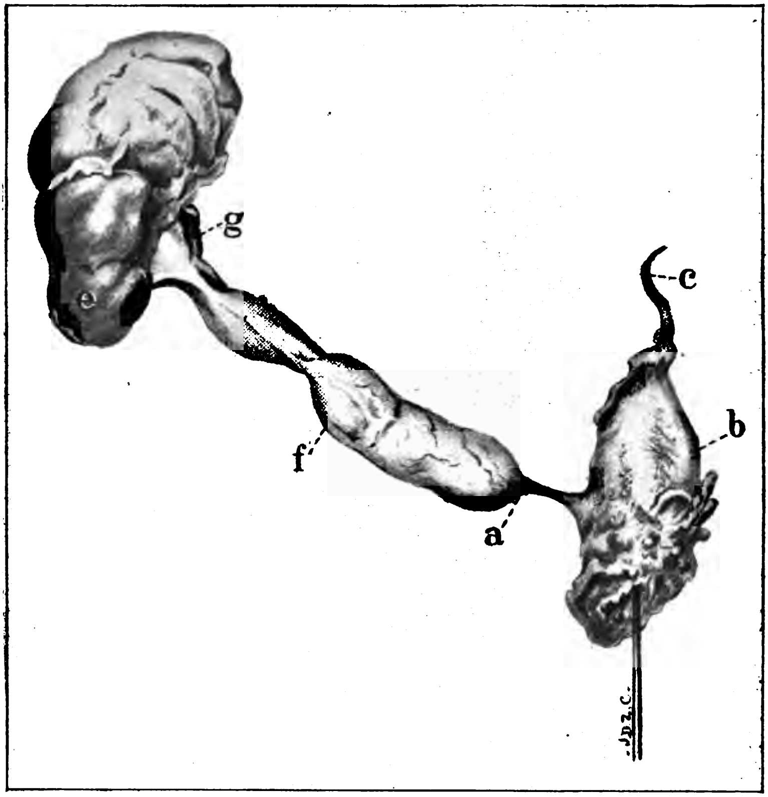
[FROM THE PATHOLOGICAL LABORATORY OF THE UNIVERSITY OF PENNSYLVANIA]

DAVID RIESMAN, M. D.

Congenital hydronephrosis is not a frequent condition, and is rarely manifested at such an early age as in the instance here reported. For this reason, and also because the primary lesion upon which the hydronephrosis depends is so obvious, the specimen possesses considerable interest. It comes from a male infant, the smaller of a pair of twins, born in the University Maternity on March 30, 1898. At birth the child weighed 3¼ pounds and presented nothing abnormal. It urinated properly and but for a persistent subnormal temperature gave no cause for anxiety until the morning of the third day, when it became cyanosed rather suddenly.

Artificial respiration seemed to revive it, but a second attack of cyanosis occurred in an hour, and in this the child died, being less than three days old.

At the autopsy nothing was found abnormal except the right kidney and its ureter. The kidney was somewhat enlarged and cystic to the touch. The pelvis was dilated and the ureter greatly lengthened and distended. It made, in situ, apparently two full, irregular, spiral turns, and ended about 1 c. m. from its point of entrance into the bladder in an impervious fibrous cord which was



CONGENITAL HYDRONEPHROSIS.

Explanation of plate—The pelvis (g) and the ureter (f) are dilated. The latter has been untwisted and the tortuosity is scarcely shown. From (a) onward the ureter is totally impervious; (b) bladder; (c) urachus.

inserted into the bladder in the usual position of the ureter. When fully distended by pressure upon the kidney the ureter had the size of a little finger. The other kidney presented no abnormality. There were no uric acid infarcts in it and presumably not in the right kidney, though the latter was not incised.

Hydronephrosis (uronephrosis—Guyon and Albarran[1]) is generally divided into the acquired and the congenital, and, further, into the total and into the partial. Englisch,[2] a score of years ago, proposed a classification into primary hydronephrosis, that due to intrinsic lesions of the urinary tract, and secondary, that due to extrinsic lesions. Hydronephrosis may be unilateral or bilateral; it may, following the terminology of Terrier and Baudouin,[3] be closed (hydronéphrose fermée), when the obstruction is total, or open (hydronéphrose ouverte), when urine can escape into the bladder, or it may be intermittent, which explains itself.

Acquired hydronephrosis is frequently met with both clinically and at autopsy. Naturally, the fundamental requisite for its production, as it is also in the case of the congenital, is some interference with the free outflow of urine, an interference that may vary in nature, position, and degree. In the first place we may mention among the causes foreign bodies in the urogenital tract, principally stones, perhaps also blood-clots, parasites, detached portions of tumors, etc. Comby[4] believed that in infants and children uratic dust and calculi might cause hydronephrosis. Four cases of this kind were reported by Bernard[5] at the meeting of the Association française d'Urologie, in 1897. One was a child eight months old with bilateral hydronephrosis and dilatation of the ureter. The urine contained in suspension a powder consisting of uric crystals. The other three were six, five, and six months old respectively. In the last the dilatation was confined to the ureter and had not yet affected the pelvis. In all uric acid crystals were found.

Then there are tumors of the tract itself and tumors and other conditions external to it and exerting pressure upon it, as, for example, tumors of the pelvis, the pregnant uterus, the retroflexed uterus, and tumors extending to the ureter or the bladder from the environment. In the case of a colored man at the Philadelphia Hospital, who came to autopsy a few days ago, a large pelvic tumor, sarcomatous in nature, had caused pressure on the bladder near the ureteral orifices and had led to double hydronephrosis. Prolapse of the uterus may also lead to the condition, and Freitag[6] states that in cases of contracted pelvis from rickets or osteomalacia the ureter may be compressed by an overfilled rectum or bladder.

Inflammatory processes in or about the ureter or the renal pelvis may produce obstruction. Tuffier[7] reports a case in which the ureter formed a sharp bend 1 c. m. from its origin by lying astride of the renal artery and vein, the bend being partially fixed by adhesions passing between its ascending and descending limbs.

Diseases of the bladder involving the ureteral orifices, enlargement of the prostate gland, and stricture of the urethra, may also lead to hydronephrosis.

A cause which clinically perhaps is the most interesting is the kinking or twisting of the ureter that sometimes occurs in floating kidney, as pointed out by Landau.[8] In this condition the hydronephrosis is usually intermittent and attended with much pain (Dietl's crisis). The degree of hydronephrosis, however, is probably overestimated. Albarran[9] has shown that the distension of the pelvis is but moderate, and that the enlargement of the kidney is to a considerable degree dependent on acute congestion. The great quantity of urine—as much as a quart—that is voided at the end of the attack after the ureter is untwisted is due to subsequent secretion, rather than to discharge of an accumulation of pre- or intra-paroxysmal origin.

The hydronephrosis of movable kidney is an open hydronephrosis, because the obstruction is due to a mere bending of the ureter, which does not cause an entire cessation of the flow of urine. The bend may, however, become permanent through peri-ureteral adhesions or other causes, the hydronephrosis thus being converted into a closed hydronephrosis (hydronéphrose fermée).

Among rare causes of hydronephrosis we may mention one pointed out by Küstner.[10] In some cases of inflammation of the pelvis the mucous membrane, which is movable over its substratum, may be drawn in front of the ureter and produce temporary occlusion by acting like a valve. A somewhat similar condition may, according to Hansemann,[11] be produced by traction on the ureter itself, whereby an angle is formed that similarly exerts a valve-like action. James[12] reported a case in which he believed the hydronephrosis was dependent on the contraction of the bladder due to the frequent micturition.

Hydronephrosis may be traumatic. The mechanism in such cases, as Wagner[13] has shown, is as follows: In consequence of the injury there is an extravasation of urine about the kidney, a so-called pseudo-hydronephrosis which compresses the ureter, or the ureter may become occluded by blood-clots or constricted by cicatrices or adhesions. Monod,[14] however, denies the occurrence of traumatic hydronephrosis, believing that in all cases there is an escape of urine into the retro-peritoneal cellular tissue, generally through a partial rupture of the ureter, rarely through a laceration of the kidney itself; in other words, the accumulation of urine takes place not in the renal pelvis and kidney, but outside.

The causes of *congenital hydronephrosis* are manifold: perhaps the most frequent is narrowing or obliteration of the ureter. This may occur at the origin, at the termination, or in the course of the tube. In 40 per cent of the cases collected by Englisch the obstruction was at the beginning. At times the occlusion is total, as in a case reported by Bonnet.[15] In rare instances the ureter is entirely absent.

Another cause is an anomalous opening of the ureter into the bladder, which occurs especially in cases of kidneys with double pelvis and double ureter. The part of the kidney drained by the ureter with the lower vesical opening becomes hydronephrotic. In any case, however, in which the mouth of one ureter is lower than that of the other, the former ureter has to traverse a longer distance in the bladder wall and is, therefore, subjected to pressure, and the corresponding kidney generally becomes the seat of hydronephrosis.

The ureter may empty into the urethra, the vagina, the intestines, or the uterus. Weigert reports a case in which a ureter emptied into the prostatic urethra. The hydronephrosis, which was partial, there being another ureter for the same kidney, was due to a sphincter-like action exerted at the mouth of the ureter by the muscles through which it passed, to prevent constant dribbling. The ureter was thus not alone a duct but also a reservoir for the urine.

At times the cause is a valve, either in the renal pelvis, similar to those described under the head of acquired hydronephrosis, or in the ureter, or even in the urethra.

Absence of the urethra or congenital phimosis (Karewski[16]) may induce hydronephrosis. Lannelongue and Achard[17] have pointed out that when the kidney is situated lower down than is normal, the beginning of the ureter comes to lie on the sacrum and may be compressed against it. In a curious case reported by Kussmaul[18], and in a similar one of Rokitansky's,[19] the ureter was compressed by an anomalous artery. Bending and kinking of the ureter may be congenital, as in a case observed by Weigert.

In horseshoe kidney, when the concavity is directed upward, the ureter is not rarely compelled to make a sharp bend to descend into the pelvis, and, therefore, hydronephrosis is occasionally found in horseshoe kidney (Brinon)[20]. Sometimes a hydronephrosis is met with in infants without the cause being apparent—probably in such cases an intermittently acting condition was at work, perhaps floating kidney, which, as Comby has shown, may be congenital, or the disease was in reality acquired through uratic deposits that were afterwards dissolved.

From the standpoint of morbid physiology the cause of hydronephrosis is either a slowly oncoming obstruction or one acting intermittently. An acute permanent obstruction, as Cohnheim,[21] Legueu,[22] and others have pointed out, very soon leads by counter-pressure to circulatory disturbances, and, finally, to suspension of urinary secretion, and in such cases the hydronephrosis is slight. Some authorities have, indeed, held that total obstruction is never followed by hydronephrosis. It has, however, been definitely proved by Albarran and Legueu that ligature of the ureter can, in dogs at least, produce a hydronephrosis, though one of moderate degree only. Atrophy of the renal tissue follows after a variable time. Open hydronephrosis, as they have also demonstrated, is slower in development, but obtains a much larger size, than one that is closed.

In cases of slowly developing or intermittent obstruction, the urinary secretion continues until the pressure in the pelvis exceeds the secretory pressure.

The kidney undergoes certain changes in hydronephrosis—the pyramids become flattened and eventually a pressure atrophy causes a disappearance of a large part of the renal tissue; that which remains becomes the seat of interstitial inflammation. In extreme cases, as in one reported by Ayrer,[23] the kidney and pelvis are converted into a huge sack, without scarcely any visible renal structure remaining. Such sacks may contain enormous quantities of fluid, at times as much as 20 or 30 liters. The capsule in these cases is apt to form adhesions to other abdominal viscera.

The fluid is at first urine, but somewhat deficient in urea. Its specific gravity varies from 1.007 to 1.012. Later the urea, phosphates, and chlorides become greatly diminished. Epithelial cells from the kidney and pelvis are found, sometimes also red blood cells. The fluid may also contain albumin, the result of secretion of the mucous membrane of the pelvis and calices. In long standing cases the fluid may become gelatinous like that of renal cysts. It may even be gruel-like and may contain crystals of cholesterin.

Hydronephrosis may be bilateral, but is more often unilateral. Roberts[24] found it unilateral in thirty-two out of fifty-two cases. In the vast majority of instances the disease is on the right side. It is more common in women than in men—of Lotheisen's[25] eleven cases seven occurred in women, while among twenty-five cases observed by Tuffier[26] twenty-one were in women.

The disease may occur at any time of life. It may be present at birth, and was no doubt so in my own case. Sometimes when of intrauterine origin it leads to the death of the fetus. At others, the urinary secretion, which is inaugurated at the fourth month of fetal life, accumulates to such an extent as to lead to the formation of a distinct tumor which may act as a hindrance during labor.

A feature that attracted my attention in looking over the literature was the existence of tuberculosis in a number of instances of hydronephrosis in children. It was probably only a coincidence. I have not had an opportunity to investigate the matter exhaustively.

It would be interesting to know to what period in fetal life the obstruction found in the case here reported dates back. Had it occurred at a time when the ureter became separated from the Wolffian duct—for a time the two empty jointly into the bladder—about the eighth week, it is not likely that the development of the kidney and ureter would have proceeded to the apparent completeness which it has actually attained. We are compelled, I take it, to consider the stricture as having arisen quite late in fetal life, after the kidney and ureter had nearly reached their full development. As to the immediate cause of the stricture, nothing definite can be asseverated. That the hydronephrosis was not greater in degree is readily explained on the ground that we have here a hydronéphrose fermée, a closed hydronephrosis, which is an obstacle to its own enlargement. The counter pressure in time reached a point where it exceeded the pressure of secretion.

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Primary Cancer of the Liver.

[FROM THE LABORATORY OF THE JEFFERSON MEDICAL COLLEGE HOSPITAL.]

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The patient, aged 55 years, was admitted to the Jefferson Hospital, in the ward of Dr. Joseph Hearn, to whom I am indebted for the clinical data, on the twenty-fourth of May, 1898. He was brought to the hospital as an emergency case. He was extremely weak, his mind hazy, his whole appearance conveying the idea of impending coma. There is a family history of malignant disease, an aunt or an uncle, or possibly both, having died from cancer, the seat of the cancerous lesion not determined. Up to six months before his admission the patient had enjoyed good health. At or about that time he suffered with an attack of severe pain in the hepatic region; the physician who saw him at this time considered the case one of hepatic colic due to gall stone. This diagnosis was further substantiated by the presence of a fairly marked jaundice. The attack passed off in a few days leaving the patient very much weakened, with a sallow skin, and with slight evidences of emaciation. The loss of flesh and increasing weakness have become more marked. A number of attacks similar to the foregoing, and associated with jaundice, have occurred from time to time. The pain has not been severe nor the jaundice marked. The total loss of weight is estimated at about twenty pounds. At the time of his admission, in addition to the nervous symptoms above mentioned, the patient showed a peculiar trembling weakness with a barely recognizable jaundice. The liver was apparently somewhat enlarged, but the gravity of the symptoms precluded an exhaustive examination, the patient being evidently in a dying condition.

With the extremely thin abdominal wall, it was possible to make out the margin of the liver with its bossed protuberances, "tubercles of Farre," justifying the diagnosis of cancer of the liver. The history of biliary colic associated with jaundice, which seemed to have been remittent rather than intermittent, would apparently have justified the diagnosis of primary cancer of the gall-bladder or the cystic duct, secondary to hepatic calculi and with metastases in the liver. On admission there developed marked shock with a temperature of 96.6° F., which, in the light of the post-mortem lesions, might be attributed to either of two conditions, (*a*) cholemia, or (*b*) interstitial hemorrhage or hemorrhage into the peritoneum in or from some of the cancerous nodules. As the patient apparently revived from the shock while the evidences of biliary poisoning became more marked, it would seem reasonable to suppose that the reduction of the temperature was secondary to the interstitial hemorrhage rather than the cholemia. The patient gradually developed an intensifying coma, in which he died May 25, 1898. There was no opportunity for examination of the urine, nor for any observation bearing upon the condition of the stools. The pulse during the patient's stay in the hospital ranged between 108 and 112; the respiration 22 to 28.

The necropsy was conducted by Dr. R. C. Rosenberger eighteen hours after the death of the patient. There was considerable prominence of the superficial veins. The skin was faintly jaundiced, while the conjunctiva showed a much more marked yellowish hue. The subcutaneous tissue did not show evidence of marked emaciation. The peritoneal cavity contained 1000 c. c. of slightly blood-tinged fluid. The intestines were distended with gas.

Thorax.—With the exception of old adhesions the pleuræ were normal. The pericardium was normal. The heart weighed 320 grams; the valves of the right side were normal. The mitral valves showed some thickening with evident shortening of the cusps. The aortic leaflets showed fenestration above the line of contact, with slight atheroma at the base of the leaflets. There was an excess of sub-pericardial fat, which at some points seemed to penetrate between the muscle fibers. The myocardium was pale and rather soft. The left lung weighed 450 grams; it showed considerable œdema and marked hypostatic congestion. The right lung weighed 480 grams, and presented the same changes.

The spleen weighed 110 grams and was apparently normal. There was no evident lesion of the adrenals. The left kidney weighed 140, the right 150 grams. The renal vein from the left kidney emerged from the upper end of the organ by three branches which united to form the normal vein. The kidney tissue cut with slightly increased resistance, the cortex was slightly thin, the pyramids congested. The capsule stripped off easily. There was no evident lesion of the stomach or duodenum.

The liver, which is here exhibited, extended slightly below the margin of the ribs; the superior surface was firmly attached to the diaphragm by fairly dense adhesions. The organ is greatly enlarged, measuring in its transverse axis 33 c. m.; in the axis of body, 25 c. m., and through its thickest portion, 16 c. m. It weighs 3900 grams. The surface everywhere shows innumerable grayish-white areas varying in size from a pin point to 2.5 c. m. Those larger than two or three m. m. are raised above the level of the surrounding liver tissue and stand out as distinct grayish nodules in contrast to the deeply congested surrounding hepatic tissue. Practically all of the nodules larger than .5 c. m. show slight umbilication, which is more marked in the larger nodules. Some of the nodules immediately under the capsule of the organ have an almost scarlet zone of redness partly surrounding them; these nodules are denser than the adjacent liver tissue, while the grayish-white nodules are as a rule less dense. The scarlet nodules on incision exude a bloody fluid and are apparently the seat of interstitial hemorrhages. Incisions into the liver show that the nodules present on the surface extend as more or less lobular masses into the organ; in addition, throughout the interior of the organ are innumerable grayish-white nodules varying in size from scarcely recognizable points to masses 3 or 4 c. m. in diameter; nearly all are fairly sharply differentiated from the surrounding tissue; at some points, however, there is a suggestion of blending, the tissue of the nodule gradually fading from its junction with the hepatic tissue toward its grayish-white centre. Many of the nodules show hemorrhagic changes similar to those noted on the surface, and, at a few points, hemorrhage can be seen in the apparently uninvolved hepatic tissue. So far as the incisions made permit of examination, it is impossible to say that any one area of the organ shows lesions older than other areas; in every part of the organ we find large and small areas. These, however, are so distributed that one is unable to say that the nodules of any particular area are older than the nodules in other parts of the organ. There is no evidence of biliary obstruction in the hepatic tissues, or in the excretory ducts.

The gall-bladder is distended with a grumous bile, which can be forced through the common duct into the duodenum. There is no dilatation of the ducts. The gall-bladder measures 10.5 c. m. in length, and 5.5 c. m. in its transverse diameter; its wall seems slightly thickened, the lining mucosa is without any evident lesion. Just where the gall-bladder terminates in its duct there is a transverse partition with a slight sacular cavity beyond it; the walls of this cavity are ribbed as though they might at some time have contained calculi; there is not, however, sufficient evidence to confirm this suggestion. There is no evidence of inflammation in or around the biliary ducts such as one would expect to find had the patient suffered from repeated attacks of hepatic colic due to the passage of gall-stones.

The pancreas weighed 110 grams, and presented no recognizable lesion. Around the head of the organ, in the retroperitoneal tissue above the pancreas, behind the stomach and also extending latterly nearly to the perirenal tissues, was a collection of nodules, apparently enlarged lymphatic glands. The largest of these was pear-shaped, 4 c. m. in its longest diameter, and situated directly by the side of the head of the pancreas, to which, however, it showed no direct connection. On incision these glands were rather firm, grayish-white in color, with occasional points of interstitial hemorrhage. There were no glandular enlargements in the lower portion of the abdominal cavity. The mesentery and omentum showed an abundant accumulation of fat, such as we commonly see in cirrhosis of the liver associated with portal obstruction. The intestines contained a small quantity of grumous, bile-stained material; there was no evident lesion of the mucosa.

Morbid Histology.—The tissues from various organs were hardened in Heidenhein's fluid, osmic acid, absolute alcohol, sectioned and stained. With the exception of slight interstitial change in the kidneys, there was nothing bearing especially upon the lesion under consideration, except in the liver and retroperitoneal lymphatic glands.

Liver.—While there is no recognizable difference in the gross characteristics of the nodules, there is a most remarkable diversity of structure under the microscope. Several distinct histologic types of the lesions can be identified. The first of these, I shall describe, is stained with hematoxylin and picric acid. The tumor mass can be readily seen with the naked eye, and is .4 c. m. in diameter. It is sharply differentiated from the surrounding liver tissue macroscopically as a rather pinkish area in the section, sharply outlined from the brownish purple adjacent liver tissue. There is the smallest possible amount of stroma which, in band-like projections, extends through the tissue, and at some points forms a pseudocapsule to the nodule. The epithelial cells which occupy the alveoli are extremely irregular in size and shape. They show at points a tendency toward a cylindrical form with an arrangement, in relation to the surrounding stroma, which suggests cylindrical-celled epithelioma, presenting a rather unusual type. The blood-vessels which traverse the stroma are few in number. The liver tissue immediately surrounding the nodule, and often some distance away from the nodule, shows widely distended capillaries, and in many areas a rather diffuse hemorrhage into the hepatic tissue. The protoplasm of the adjacent liver cells is slightly granular, while the crowded irregular cells manifest the results of evident pressure. A few of the liver cells show fragmentation, and occasionally one notes the presence of massive fat globules. In addition the liver tissue shows an extensive infiltration by leucocytes; these are, for the most part, distributed diffusely throughout the structure, although at points there is evident agmination into groups of round cells containing but few liver cells in the area involved. Differential staining proves these cells to be, for the most part, mononuclear, although occasionally polynuclear cells are found. Roughly and superficially observed, the nodules shown in this slide suggest, in a very unusual way, the structure of a salivary gland and, though possibly less characteristically, the pancreas. It does seem possible to say exactly in what part of the hepatic tissue nodules belonging to this group began; the small quantity of connective tissue, the absence of bile-ducts and the limited number of recognizable blood-vessels, might lead one to infer that these masses originated within the lobule.

Another type of lesion to be recognized is distinctly that of the scirrhous form of carcinoma. The most characteristic areas are manifestly in the connective tissues between the lobules, as is shown by the presence of apparently uninvolved bile-ducts in the midst of tissue which is structurally scirrhous carcinoma. The alveoli are loosely occupied by epithelial cells possessing the usual characteristics noted in scirrhous carcinoma of the mamma. The liver tissue adjacent to these areas shows advanced necrotic changes. In many areas the protoplasm of the hepatic cell refuses to take the stain, and is recognizable under the microscope as a more or less hyaline or granular zone, irregular in thickness and but loosely connected with the faintly stained nucleus. Many of the nuclei do not stain at all. In addition to the necrotic and degenerated condition of the liver cells in these areas, there is an abundant presence of red blood-cells, some of them apparently normal, and others in various stages of fragmentation. With the enormous number of red blood-cells present one would expect the reaction of fibrin; careful staining, however, fails to reveal the presence of that body. As is to be expected in hepatic tissue showing the histological characteristics of scirrhous carcinoma, many areas have large alveoli with a small amount of stroma, with closely packed and rapidly growing epithelial cells, such as one commonly sees in encephaloid cancer. These areas are more marked in the larger nodules; indeed the larger nodules on the surface, showing central softening with umbilication, are almost exclusively made up of tissue closely resembling the ordinary encephaloid cancer. Within these encephaloid areas one notes abundant widely diffused hemorrhages. As a rule, neither the scirrhous-like nodules nor the encephaloid masses shows the sharp limitation noted in the first type of the growth. Sections stained in alkaline toluidin blue show the scirrhous and encephaloid areas best, while sections stained by Pianese's carmine and picro-nigrosin differentiating stain, the nigrosin having been replaced by Indulin, show differentiation between the stroma and the alveolar contents. Sections stained with alkaline toluidin blue and treated with ferrocyanide and tannic acid show beautifully the degenerative changes present in the liver tissue. Sections stained with alkaline toluidin blue and eosin show well the hemorrhagic infiltration.

The retroperitoneal glands show the histologic structure commonly observed in the secondary metastases of scirrhous and encephaloid carcinomata.

In some particulars the case presents points of unusual interest, bearing out the statement made by DaCosta (Medical Diagnosis, seventh edition, page 621), that primary cancer of the liver usually is associated with the history of cancer in the family. This patient had an aunt and possibly an uncle die of cancer. In common with the observations of Frerichs, Merchison and Hanot this organ was very much enlarged. Unlike the observations of the authors mentioned and other writers on the subject, this organ shows no large primary lesion with secondary metastases, but possesses the appearance of a widely diffused primary lesion. The organ here presented does not possess the characteristics of the massive form or the "cancer avec cirrhose" or the interlobular form of cancer commonly described.

To those who believe in the unity of the various forms ot cancer, this specimen presents confirmatory evidence. We have not been able to find reference to the occurrence in the liver of cylindrical-celled adeno-carcinoma associated with the medullary type of cancer.

As indicating the possible lymphatic channels through which the liver communicates with the general lymphatic system, the enlargement of the glands distributed above the pancreas and in the retroperitoneal tissues may be of more than passing interest to the anatomist, who, to a certain extent, must depend upon such morbid states to demonstrate lymphatic connections not demonstrable by ordinary injection or dissection methods. *June 23, 1898.*

Diffuse Tuberculosis of the Testicle.

[FROM THE LABORATORY OF THE JEFFERSON MEDICAL COLLEGE HOSPITAL.]

W. M. L. COPLIN, M. D.

The patient, aged 60, has nothing in his individual history bearing upon the organ in question. There is no collateral history of tuberculosis. Mother died of heart disease. One sister living and well. Three brothers died during infancy.

The present trouble dates from March of this year (1898), when he received a blow in the right groin, which was followed by the development of a hydrocele which assumed its maximum size in twenty-four hours. The condition was not associated with pain, but with a sense of weight and dragging with slight evidences of tension. Under treatment the hydrocele disappeared. Four weeks later, without any assignable cause, symptoms set in which suggested strangulation of the testicle, associated with some pain and the redevelopment of the hydrocele with symptoms similar to those noted before. The hydrocele was aspirated, and 250 c.c. of clear straw-colored fluid, such as is ordinarily found in hydroceles, was drawn off. On the 13th of May, 1898, the patient was admitted to the Jefferson Medical College Hospital, under the care of Dr. Orville Horwitz. One week prior to admission the hydrocele had redeveloped, but only to a slight extent.

On admission the right testicle was enlarged, hard and indurated, slightly painful on pressure, and during walking. There was a more or less constant sensation of dragging. The temperature was 98.2° F., pulse 70, and respiration 17. The urine was acid in reaction, had a sp. gr. of 1020 and contained no albumin or sugar. There was no evidence of vesical complication or of prostatic enlargement. The general health of the patient was fair, although he had lost some weight. On May 14, Dr. Horwitz removed the testicle. The opposite organ was apparently normal and was not disturbed. Since the operation the patient has gained in weight and enjoys perfect health.

The specimen as received at the laboratory consisted of the testicle, epididymus and vas deferens all covered by their proper tunics. The mass weighs 117 grams, and is here presented.

The testicle is laid open in its long diameter, from the tunica albuginea down to and partly through the mediastinum. The measurements are as follows: Length 8 c. m., width from the mediastinum to the tunica albuginea, 5 c. m., transverse measurement, 4.2 c. m. The general contour of the organ has been retained. The incised surface bulges forward, is reddish gray in color with distinct yellowish gray dots which closely resemble tubercles. These vary greatly in size and outline; the smaller points are approximately 1 m. m. in diameter, perfectly round and surrounded by a grayish translucent almost uniform matrix. The larger tubercle-like areas measure as much as .5 c. m. They are distinctly yellowish in the center and a few of them are centrally caseous. The areas are irregularly distributed as to size, larger and smaller areas occurring side by side. They are arranged, however, radially from the mediastinum to the periphery, fan-like in their areas of distribution, the petal-like blades of the fan having their widest point at the surface just under the tunica albuginea. None of these masses can be made out in or on the tunica albuginea, although its surface is roughened and at points a barely discernible fibrinous covering can be detached. The tunica albuginea and tunica vasculosa appear to be thickened. The fibrous septa are hyaline, apparently œdematous with punctate areas of what appears to be hemorrhage or widely distended blood-vessels. In most of the organ the tubercle-like points seem to be restricted to the lobules; there are, however, areas in which it is impossible to say that the septa are not involved, indeed they appear to contain the tubercle-like nodules. The mediastinum is swollen, œdematous, hyaline and shows a translucent grayish surface scarcely resembling the normal. There are no recognizable tubercles in this structure. The whole organ is much enlarged, the increased volume seems to be more in the areas of the lobules than elsewhere, although all the structures show considerable swelling. The tissue cuts with marked resistance, and to palpation seems much denser than normal.

The epididymus is very much enlarged; it projects slightly at both ends of the greatly enlarged testicle. The coni vasculosi and the globus minor form one immense, dense, cord-like body, cylindric in shape, 9 2 c. m. in length and 1.6 c. m. in diameter; it possesses a wood-like denseness and cuts with great resistance. There are innumerable tubercle-like dots disseminated throughout the entire mass, and many small abscess-like areas which contain a cream-like pus rather than caseous material; in only two of these areas do the contents at all resemble the caseous material commonly found in tuberculous abscesses, improperly so-called. The entire cylinder, presumably once composed of differentiable structures, the globus minor, body and the globus major, can no longer be separated into dissimilar tissues. The matrix is uniform from one end of the organ to the other, hyaline, grayish, semi-translucent and occupied throughout by tubercle-like areas, except for the occurrence of abscess areas, closely resembling those already described in the lobules.

The vas deferens is thickened, measuring at points .4 c. m. in diameter. The thickening is not uniform, showing at points as a fusiform enlargement. As it approaches the point of separation from the stump it becomes uniform in diameter, and measures a scant .25 c. m. On section a small quantity of cream-like material exudes.

Microscopic examination of the fresh pus from the interior of the abscess-like areas in the epididymus shows granular detritis, fat in abundance, and a few leukocytes, the latter granular, approximately pus cells. In addition to the foregoing are granular masses resembling tube-casts from the kidneys. It seems not improbable that these are from the straight tubules of the testicles.

The blocks were hardened in corrosive sublimate, infiltrated with paraffin, sectioned, stained with carmine, hematoxylin and eosin, hematoxylin and picric acid, and with various aniline dyes, including stains for tubercle bacilli, stains by Gram's method, and other bacterial stains.

Sections taken from the periphery of the testicle show practically no tissue which would enable one to distinctly identify the organ. The remaining constituents which can be recognized are the tunics and their fibrous extensions into the organ, constituting the septa. The tunics which cover the organ are bound together and cannot be differentiated at all points. The tunica albuginea and tunica vasculosa are very much thickened; the thickening is more marked in the tunica albuginea, which shows a productive inflammatory process on its surface, the younger cells being external, the older, nearly fibroid areas, lying next to what appears to have been the normal membrane. At points there appears to be a productive inflammatory process in progress between the tunica albuginea and the tunica vasculosa. In many areas, however, the two tunics can not be histologically differentiated. In the tunica vasculosa there is little round-cell infiltration except immediately adjacent to the lobules and, to a less marked degree, at the points from which the septa are projected inward.

The convoluted tubules present in the lobules are not distinctly identifiable. Knowing the structure with which we have to deal, one quite readily recognizes what were probably, at one time, parts of the convoluted tubules, but if one did not know that the tissue under examination was testicle, it would be impossible to say that the masses seen under the microscope were part of the convoluted tubules. The convoluted tubes are filled with cellular detritis, many of the cells showing fragmentation and occasionally closely resembling, in the center of the tube, the appearance seen in a tubercle in which degenerative changes are just beginning to evince themselves. Just around these central degenerative masses are numerous large mononucleated cells, probably the progeny of the epithelial cells which line the tubules. External to this, in the area normally occupied by the flattened connective tissue cells, we find its structures infiltrated by an abundant collection of mononuclear, round cells, with a scant perinuclear protoplasm. In the interstitial portion we find scarcely any areas which can be said to approach the normal. In this part of the organ are innumerable tubercles in all possible stages of development, and showing all degrees of degenerative changes. Some of the tubercles show merely as beginning aggregations of mononuclear cells, the others show in addition the characteristic external layer with beginning central softening, while the larger masses are seen to contain well-marked caseous areas. I do not recall having examined any tuberculous tissue which contained anything like the number of giant cells which are present in this structure. Many of the blood-vessels show well-marked endarteritis. The inflammatory condition is not of the same age in all parts of the organ, some of the blood-vessels showing an organized thickening of the intima, while in others the cell accumulation is still embryonic in character. I am not fully satisfied that it is possible to identify in the interstitial structure the interstitial cells presumed to be characteristic of this organ.

While microscopically the septa did not show any evidence of tubercles, under the microscope many beginning tubercles are recognized. Aside from the invasion by tubercles there is nothing deserving of special mention. Bacterial stains made from this part of the organ demonstrated only a scant number of tubercle bacilli. These are present in the spreads made from the cut surface and also in the sections. Small areas with more recent invasion show a rather generous number of tubercle bacilli; they are not, however, evenly distributed. When found they are clearly and distinctly demonstrable.

The sections taken from the central portion of the testicle including the inner part of the lobule, the straight tubules and mediastinum with the rete testis show nothing different from the periphery of the organ except that the tubercles are less abundant in the mediastinum. Some disorganization of tissue with coagulative hyaline and caseous degenerative processes is irregularly disseminated throughout these areas. The mediastinum may be said to show the changes less than any other part of the organ, although an abundance of hyaline material is demonstrable throughout its structure.

The evidence to be gathered from the examination of the epididymus indicates that the lesion was in all probability primary in this structure. Like the testicle proper there is little of the normal tissue which can be identified. At but few points can one find evidence of the tube of the epididymus. The tuberculous areas are larger and the accompanying degenerative changes more marked than in any other part examined. The whole tissue is literally packed with tubercles in all stages of development and degeneration. The matrix between the tubercles is hyaline at points and everywhere infiltrated with round cells. Most of the new cells present in this matrix are in the embryonal stage and but little attempt at fibrous change can be found. The invasion by leukocytes seems to be unusually marked. The caseous, coagulation, and hyaline changes are extensive. Cover-glass spreads made from this area show numerous tubercle bacilli and staphylococci. The tubercle bacilli were readily stained in the sections as were the staphylococci.

The wall of the vas deferens is at points thickened, at other points it is considerably thinner than normal. From the flattened condition of the epithelium and the distended character of the tube, the thin points are presumably due to distention. For the most part there is little change in the muscular fibre of the tube, but in the submucosa there is an unusual amount of round cells, while the epithelium is, in many areas, desquamated and has almost disappeared; at other points it is fairly retained and occasionally shows its columnar character. The blood-vessels in the submucosa are intensely engorged, and at a number of points have the appearance of being actually thrombosed. In no place in the wall of the vas have I been able to demonstrate the presence of tubercles, nor did the smear preparations from the vas show tubercle bacilli.

In some of the blood-vessels in the cord there is a small amount ot endarterial change. For the most part this is limited to slight proliferative changes in the intima and in the adjacent tissue, in most instances not having gone on to fibroid changes. No thrombosis of the blood-vessels is recognizable, at least the blood within the vessels shows no degenerative change which would indicate thrombosis of any standing. Occasionally around the blood-vessels there is a small hemorrhage, the appearance of which would indicate that probably this was incidental to the ligation of the cord, as it is manifestly recent. No tubercles were found in this structure.

With the exception of the lungs we do not expect organs to show the local diffusion of miliary tubercles without a more or less general distribution of the infection. So far as I have been able to determine attention has not been called to this peculiar lesion in the testicle. The explanation is readily found in the peculiar distribution of the blood supply to the organ. The blood-vessels, branches of the spermatic artery, anastomose as a vascular net-work in the mediastinum extending into the vascular tunic of the albuginea from which they are projected into the septa between the lobules. The distribution to the epididymus is similar although not identical. When considering, therefore, the cause for the peculiar universal distribution of the lesion, in this case, one has but to conceive that a primary caseous focus infected the mediastinum either by its direct location there, or spread to this part of the organ from an earlier infection in the epididymus. Once the tuberculous lesion was thoroughly established thrombosis of the blood-vessels of the area involved and the subsequent breaking up of such thrombi containing tubercle bacilli led to the wide distribution of the lesion throughout the organ.

Discussion.

Dr. Williams recalled a case with a large central cavity, containing many tubercle bacilli.

Dr. Swan recalled a case with complete obliteration of the testicular substance.

Dr. Coplin.—Local testicular tuberculosis is not uncommon; this diffuse form however is very rare.

June 23, 1898.

A Contribution to the Pathology of the Choroid Plexus.

[From the Pepper Laboratory of Clinical Medicine.]

JOSEPH SAILER, M. D.

Through the kindness of Dr. MacNeill, of the Veterinary Department of the University of Pennsylvania, I recently obtained possession of a very interesting tumor of the choroid plexus. It was removed from a horse that died of meningo-myelitis, and the clinical diagnosis was confirmed by the microscopical examination of the brain and cord, which showed a round-cell infiltration in the pia and arachnoid. The tumor was not suspected during life. In the left ventricle there was found a mass about 4 c. m. long and 0.5 c. m. in diameter, irregularly cylindrical in shape, pale, firm, with a faintly granular surface. The choroid plexus, upon the opposite side, was apparently normal. The tumor-like mass was in no place adherent to the ependyma. In fact, when I obtained the specimen, it did not appear to be continuous with the choroid plexus, but this was probably due to separation during removal. Upon cutting into it with a knife, a slight gritty sensation could be detected, indicating the presence of calcareous material. The surface of the section was of a homogeneous grayish yellow color, with here and there reddish points. Microscopically, the appearance varies with the part from which the section is taken. The centre of the growth consists of trabeculæ of fibrous connective tissue enclosing irregular spaces filled with an homogeneous or slightly fibrillar substance; the fibrillæ occasionally staining pink with thionin. Here and there, in these homogeneous masses, could be seen collections of strongly basophilic or cyanophilic granules that probably represent degenerated nuclei. It is difficult to explain the nature of these masses, but from the fact that the spaces in which they occur bear a close resemblance to dilated venules, I am inclined to consider them as partially organized thrombi. In the midst of these masses were some collections of cholestrin plates. These do not exhibit the characteristic rhomboidal shape with the broken corner, but form somewhat irregular masses, faintly yellowish in color, and glistening brightly when examined by oblique illumination. Here and there, small dark granules could be observed that stained a dirty purplish color by hematoxylin, probably the calcareous granules detected in cutting. Toward the surface there was a collection of blood-vessels, probably venules, with greatly thickened walls. This hypertrophy of the wall may have been due to the chronic inflammation which probably existed in the plexus, but the horse was old, and it may have been, possibly, an arterio-sclerotic change. At the anterior end of the tumor, extending backward along one side, there was a minute villous-like outgrowth, pale in color, and very firm in consistency. Microscopically, this was found to consist of minute papillæ, composed of flat or polygonal epithelial cells, arranged in irregular layers and springing from a basement membrane that in some cases appeared to be perforated; at least epithelial cells were found in the fibrous substance beneath it. This growth resembles very closely an ordinary papilloma of the skin, and belongs to the type known as papillary epithelioma, a tumor that has already been described as originating from the choroid plexus. The specimen is interesting, inasmuch as it illustrates a great variety of the pathological processes that occur in the choroid plexus. This, as is known, is formed by the invasion of the primary ventricles of the brain during embryonal existence by a mass of blood-vessels. These push before them the roof of the neural canal, which forms an epithelial layer closely applied to all of their finer ramifications, and ultimately the choroid plexus may be said to consist of a tube lined by endothelium and covered by epithelium, with a small amount of delicate fibrous areolar tissue between the two layers. This condition is most distinct in some of the lower animals. Dr. A. E. Taylor recently very kindly placed at my disposal the brain of an amphiuma. In this animal the choroid plexus, in the lateral ventricles, consists of a single blood-vessel covered by epithelium, showing the elementary structure very clearly, particularly as the nucleated blood cells in the lumen of the vessel are distinct. The epithelial cells are cuboidal and not columnar in shape, and I have been unable to detect any trace of cilia. It is, of course, obvious that the choroid plexus can have no nutritive function excepting in so far as it supplies the epithelium that covers it, and on account of its close analogy in structure to the glomerulus of the kidney, it has been supposed that its object was to secrete the cerebro-spinal fluid. Whether the cerebro-spinal fluid, itself, has nutritive functions or not, it is of course impossible to say. The pathological changes found in the choroid plexus are, on account of the presence of both mesoblastic and epiblastic tissues, very numerous. Calcareous and pigmentary infiltration, the latter especially in malaria. Infiltration with cholestrin also occurs. The walls of the blood-vessels may exhibit hyaline or amyloid degeneration, or arterio-sclerotic changes. Thrombosis also probably occurs, although I have only the observation upon the specimen presented this evening to support this statement. Embolism should be certainly exceedingly rare. Hemorrhagic infiltration may occur as an agonal manifestation, or in the course of hemorrhagic diseases, or in severe inflammatory conditions. A bleeding from the choroid plexus into the ventricle, as far as I know, has never been detected. Inflammatory processes are comparatively rare. In various forms of meningitis and ependymitis, round-cell infiltration or even small focal abcesses may be detected. Tuberculosis is sometimes observed, especially in the form of miliary tubercles in cases of tubercular meningitis. Cystic degeneration is exceedingly common, and may be said to be almost physiological in old age. The cysts may be lined with endothelium and contain a clear liquid, representing dilated lymphatics. In other cases they are lined by epithelium and the contents may be a clear liquid or a rather viscid colloid mass. Parasitic cysts, particularly those due to the embryo of the tenia soleum, are also found. If single, they are spoken of as the cysticercus cellulosa, and if multiple, as the cysticercus racemosus. These cysts may occasionally become detached from the plexus and lie free in the ventricle. Of the benign tumors, fibroma, possibly organized thrombi, and lipoma may be mentioned. The latter is exceedingly rare and practically always associated with multiple lipomata in other parts of the brain. Endotheliomata spring from the lymphatic spaces as firm, white infiltrating tumors, exhibiting, microscopically, the characteristic alveolar structure. Two forms of epithelioma have been described. The papillary epithelioma, that is, the form that I have exhibited this evening, which is probably non-malignant; and the so called cylindrical carcinoma of the ventricle, a growth composed of columnar cells, that usually pierces the ependyma and involves the brain substance. This is malignant by position, but rarely gives metastasis, and there is some doubt as to its nature and whether it actually springs from the cuboidal cells of the choroid plexus, or from the ependyma lining the ventricle.

June 23, 1898.

A Tumor Resembling in Structure the Choroid Plexus.

W. G. SPILLER, M. D.

Dr. Spiller showed a vascular tumor which resembled in its structure the choroid plexus. He was not certain that the sections had been taken from an area remote from the plexus, but was under the impression that they had been. The various processes were composed almost entirely of cuboidal and somewhat cylindrical cells, arranged in such a manner that they formed a central lumen containing blood. Unlike the choroid plexus, however, these processes were intimately connected with the white matter of the brain, and surrounding them were cells, mostly of an elongated shape, resembling sarcoma cells. The tumor was evidently a sarcoma in which the vessels had assumed a form resembling the choroid plexus. He was indebted to Drs. Sinkler, Keen and Burr for the specimen.

DISCUSSION.

DR. RIESMAN.—Dr. Spiller kindly gave me an opportunity of seeing these sections some time ago. The tumor appears to me to be what is called a perithelioma, *i. e.*, a tumor derived from the perithelium of the blood-vessels. They are not peculiar to the choroid plexus; two almost identical with this one histologically have been described by Besold; they were additionally interesting because occurring in two sisters. The cells in Dr. Spiller's case seemed to me to be, many of them, columnar in shape and to surround the vessels somewhat radially—these peculiarities have been noted in peritheliomatous tumors. These tumors, I may add, belong to the sarcomata, and are in reality endotheliomata.

June 23, 1898.

On Amyloid, Colloid, Hyaloid and Granular Bodies in the Central Nervous System.

[FROM THE WILLIAM PEPPER CLINICAL LABORATORY.]

WILLIAM G. SPILLER, M. D.

Last year I had the privilege, through the kindness of Dr. Lloyd, of presenting to this society specimens from the spinal cord of a man of forty years of age who died from a disease resembling Landry's paralysis. I was especially impressed by the great number of amyloid bodies in the spinal cord of a person of forty years. It is true that these bodies begin to be quite numerous at this age and increase in number as life advances. They are indicative of the senile changes which occur in every central nervous system at a certain period, and they are especially abundant in the olfactory tracts of man. The person to whom I refer had arterio-sclerosis of the vessels of the central nervous system in a high degree, and we may, perhaps, look upon this as a possible cause of the great number of amyloid bodies present in this case, for it has been said that "a man is as old as his arteries." We do not regard these bodies as a sign of great value in acute degenerative processes, but possibly we do not recognize their full significance. They were found in such large numbers in this case that they seemed to be one of the signs of disease of the spinal cord. They were present within the posterior columns, within the septa, about the vessels and the central canal, and especially within the zones of Lissauer. They extended also a short distance into the posterior roots.

Redlich believes that these bodies are derived from the neuroglia cells, but Homén[1] states that in the spinal cord of a dog, in which he had cut nerve fibres, he was able to find a few amyloid bodies within the degenerated areas twenty days after the operation. He found these not only fully formed, but was able also to observe intermediate stages between these bodies and altered nerve fibres. Homén believes, therefore, that the amyloid bodies are derived from degenerated nerve fibres.

The findings of Karl and Gustaf Petrén[2] are of much interest. These investigators observed numerous amyloid bodies in the spinal cord of a human fœtus. They refer to the fact that Redlich has never been able to find them in the nervous system of persons younger than eighteen years of age, and they have shown, therefore, that their development may be intrauterine.

The writer has been able to study these bodies within the central nervous system in a great many cases, and has found that they always present the same appearances. They stain a light purple with Delafield's hematoxylin and a reddish-brown with Lugol's solution, which changes to a purplish color on the addition of a little sulphuric acid. They are not exactly the same as the amyloid substance found elsewhere in the body, or as the amyloid bodies of the prostate gland.

Dr. Dercum and the writer, in studying a very typical case of amyotrophic lateral sclerosis, have found formations which resemble quite closely the amyloid bodies, and yet differ from them in important respects. They are larger than some of the amyloid bodies, but are in general round, and when deeply stained are homogeneous. When faintly stained some of them are seen to have a pale central core surrounded by a deeply stained circle, and this in turn is surrounded by a thicker circle of the same shade as the central core. They do not stain with Delafield's hematoxylin, methyl green or acid fuchsin. With gentian violet they are the same color as the surrounding tissue, but may be detected. They are a deep purple when thionin is employed, but on exposure to light the color soon fades and they become invisible. The tissue in which they are found has been hardened in formalin and alcohol.

These bodies are especially numerous within the medulla oblongata, and within the perivascular spaces, but they extend also into the surrounding tissue. We were at first almost inclined to look upon them as artifacts, but the existence of three rings within some of them, and the fact that they may also be detected with gentian violet and Lugol's solution, although staining like the surrounding tissue, show that this view is not tenable.

They differ from the hyaloid bodies in their reactions to stains; in the number and appearance of their circles, although most of them are homogeneous; in their globular form and more nearly equal size; and in the absence of a tendency to form irregular masses, except when the tissue has been kept some time in the hardening fluid. When Lugol's solution is employed they stain yellow, while the amyloid bodies are reddish-brown.

W. Bevan Lewis[3] speaks of peculiar formations under the name of colloid degeneration. These are minute, round or oval bodies, from six to twelve microns up to forty microns in diameter, and are frequently found in the central nervous system of the insane. They are supposed by him to be derived from the medullated nerve fibres. Bevan Lewis acknowledges that the name is unfortunate, for it indicates a colloid transformation of neuroglia cells similar to that of epithelial cells, and these bodies are not derived from neuroglia cells. They are spherical, ovoid, or pyriform, and in later stages may also be crenulated, a condition which we also have found. They are homogeneous, devoid of concentric markings, colorless and pellucid. They may be slightly tinged by hematoxylin, but not by carmine or aniline dyes, and they exhibit no reaction with iodine and sulphuric acid. Bevan Lewis observed these bodies in great numbers in a case of bulbar paralysis. They are usually found only in the white matter.

We believe that the bodies found in our case of amyotrophic lateral sclerosis, with bulbar symptoms, resemble more closely the colloid bodies of Bevan Lewis than any others with which we are familiar.

In a case of tumor of the base of the brain, in which symptoms of akromegaly had been noted during life, I have found the hyaloid bodies recently mentioned by Dagonet.[4] I am indebted to Drs. F. A. Packard and H. W. Cattell for the pathological material, but shall at present refer to it only in so far as concerns the bodies under consideration.

These are most irregular in shape, and many of them show a concentric arrangement. They vary greatly in size, and in some places form quite large masses. They have little resemblance to the formations previously described, and could hardly be mistaken for them. With Weigert's hematoxylin stain, they are brown; with eosin, pink; with thionin, purple; with carmine, a very pale pink; with Delafield's hematoxylin, deep purple; with acid fuchsin, red; with Weigert's fibrin stain, the color of the surrounding tissue; with iodine, yellow like the surrounding tissue; with gentian violet, purple with a tinge of pink; with Van Gieson's stain, deep reddish-brown. Boiling water, acid and alkali do not dissolve them.

They not infrequently have deeply-stained edges and pale centres, and each seems to be surrounded by a space very much like that found about the vessels. They are situated in the cortex, and form large masses in the much thickened ependyma. Not infrequently they are elongated and present much the appearance of thickened vessels, a resemblance which is increased by the space about each of them, often extending a short distance beyond them.

Edsall and Sailer[5] have recently spoken of bodies very similar to these, and have referred to the literature bearing on the subject. Since Adler described them in 1875, comparatively little, according to Dagonet has been written concerning them. Careful study in this and other cases has led me, contrary to views originally held, to conclude that probably some of these bodies are thickened vessels.

The spaces surrounding them, the concentric rings much like those I have found in diseased vessels elsewhere, the elongated form of certain of these structures, with surrounding spaces extending further than the hyaloid masses, are very suggestive of diseased vessels. Alzheimer[6] also found colloid masses separate from the vessels, and believed that they were deposited from the fluid within the tissues. He showed that the chemical reactions of the colloid degeneration of the cerebral vessels is very different in the reported cases, but he does not believe that the degeneration in these cases is essentially different. He states that he regards the possibility of glia or nerve cells undergoing colloid degeneration as very doubtful, although the cells of the vessels may do so.

The granular bodies, the *corps granuleux* of the French, the *Fettkörnchenzellen* of the Germans, are very different structures from those previously mentioned, but like them they are indicative of degeneration. Indeed, in former days, when the microscopical technique was less perfect than at present, their presence was regarded as of great value in determining the existence of degenerative processes.

These bodies may be seen in various diseases of the central nervous system, and I have found them in large numbers within the spinal cord a few days after fracture of the spinal column. I have seen them in masses, crowded together close upon one another, in processes of long duration. In old hemiplegia I have found them, when the method of Marchi was employed, filling the perivascular spaces and leaving the rest of the cord free, as far as could be determined by this method.

When examined in the fresh state they are much larger than when a hardening fluid has been employed, and are filled with fat drops.

When they wander into the nervous tissues they seem to be unable to find their way out again. They are quite large cells, and are very numerous in the brain of the newborn, and for this reason it has been thought that they may be instrumental in carrying material for the formation of the medullary sheaths (Obersteiner[7]).

They are very numerous in all acute forms of degeneration when the nerve fibres are affected, and may be shown, by the method of Marchi, to be filled with a substance resembling fat. It is believed that they are formed by fatty degeneration of nerve cells, connective tissue cells (neuroglia?) and even from the smooth muscular fibres of the vessels (Huguenin, cited by Obersteiner). Guizzetti[8] states that these cells are derived partly from wandering leukocytes, partly from transformed endothelial elements of the perivascular lymph sheaths; that they multiply by karyokinesis; and that the ganglion cells and neuroglia cells do not take part in their formation.

I have recently had the opportunity to study vessels in a condition of arterio-sclerosis from the region of the pons, and have found large accumulations of these cells within the vascular walls. They are filled with drops of fat, as shown by the osmic acid. They have usually only one round or somewhat elongated nucleus, but occasionally two nuclei may be found. They form granular masses in certain parts of the vascular walls in which the individual cells can be seen only with the greatest difficulty.

(1) Redlich, Jahrbücher für Psychiatrie, Vol. x, Abstract in Neurologisches Centralblatt, 1891. Homén, Atlas der pathologischen Histologie des Nervensystems, No. vi, pp. 12 and 18. (2) Karl and Gustaf Petrén, Virchow's Archiv. Vol. 151, No. 2. (3) Lewis, A Text-Book of Mental Diseases, p. 465. (4) Dagonet, Système Nerveux Central, Coupes Histologiques Photographiées. (5) Edsall and Sailer, Proceedings of the Pathological Society of Philadelphia, Vol. i, No. 4. (6) Alzheimer, Archiv für Psychiatrie, Vol. xxx, No. 1. (7) Obersteiner, Anleitung beim Studium des Baues der nervösen Centralorgane. (8) Guizzetti, Abstract in Neurologisches Centralblatt, No. 5, 1898, p. 211.

Sections from the Sternocleidomastoid Muscle in a Case of Congenital Torticollis.

[From the William Pepper Clinical Laboratory.]

WILLIAM G. SPILLER, M. D.

Through the kindness of Dr. Keen I am able to exhibit to you portions of muscle removed by him from the sternocleidomastoid muscle in a case of wry neck, supposed to have been congenital. The boy was seventeen years old, and since earliest infancy the right sternocleidomastoid muscle had been so much contractured that the chin was drawn very markedly to the left and upward. An incision was made parallel with the clavicle, and 2 c. m. of the lower portion of the affected muscle were removed. Cicatricial contraction followed the operation for a time, but by constant massage and movement of the head, the benefit from the operation has been such that the head is now straight. The portions of muscle removed were given to me for microscopical examination.

Erb[1], in his well-known paper on dystrophia muscularis progressiva, describes abnormalities found in that disease like those existing in this case of congenital torticollis. He found some of the muscular fibres hypertrophied and some atrophied; the muscular fibres in transverse sections were rounded and had lost their sharp angles, and some were perfectly circular. The nuclei of the muscular fibres were much more numerous, and some were situated centrally within the fibres. He found long chains of muscular nuclei, as many as twenty to forty in some fibres. He spoke of longitudinal segmentation of the muscular fibres, so that one fibre formed two or three separate parts at one of its ends. He observed vacuolation of the muscular fibres, increase of connective tissue, accumulations of connective tissue nuclei, and much lipomatous tissue. Erb believed that the multiplication of the muscular nuclei and the segmentation of the muscular fibres indicated excessive nutrition.

The changes which Erb found in progressive muscular dystrophy have been seen about tumors situated within muscles (Schäffer), and in regenerating muscles after injuries, burns, etc. (Nauwerck). Hitzig found all these abnormalities of the muscles in infantile spinal paralysis (cited by Erb).

In this case of congenital torticollis I have found considerable increase of the connective tissue and of the connective tissue cells. The muscular fibres in some places are much fewer than normal, and are circular in transverse section, and not angular; they contain many nuclei, and some also many centrally located nuclei, each surrounded by a clear space. Some of the muscular fibres are distinctly forked in longitudinal sections, so as to terminate in two or three distinct fibres, each transversely and longitudinally striated and containing nuclei. Other muscular fibres in transverse sections are found separated into two to five distinct divisions. Long chains of rectangular nuclei are found in longitudinal fibres, and sometimes two or three chains are present within a fibre. These nuclei are shorter than most muscular nuclei, are arranged at equal distances from one another, and may number twenty or more in a fibre. The appearance presented by these long chains of regular nuclei is most striking. Many of the fibres have undergone hyaline degeneration.

The situation of nuclei within the centre of muscular fibres is abnormal. Koelliker[2] says that the round or elongated nuclei in man, except in the heart muscle, are situated on the inner surface of the sarcolemma.

Golgi[3] observed nuclei within the muscular fibres in adult man, but he regarded them as very different from the sarcolemma nuclei. They were especially numerous near the tendons, while rare in the remainder of the muscular substance.

Dejerine and Sottas[4] observed centrally situated muscular nuclei, as well as the long nuclear chains, within the fibres in the only case of Thomsen's disease which has ever been examined postmortem. I have sections from this case, but the pictures published with their report are sufficient, and show the resemblance of the muscles in Thomsen's disease to those in progressive muscular dystrophy.

Morpurgo and Bindi[5] have studied the muscles of an eight months' human fœtus and of adult man. The muscular fibres are of more equal size and smaller in the fœtus, and the number of nuclei in the different fibres is nearly the same. In adult human muscles of fine calibre (the external muscles of the eyes, the zygomaticus major), the nuclei in the smaller fibres are about as numerous as in the fibres of the same calibre in the fœtus, but as the fibres are longer, the total number of nuclei in each fibre must also be greater.

The smaller the fibre the thicker are the nuclei, and the latter become less numerous with the growth of the muscular fibre. In hypertrophy due to activity the small fibres grow more rapidly than the large, and their relative excess of nuclei is indicative of reserved growth force.

It may be, therefore, that the many nuclei found within the muscular fibres in this case of congenital torticollis are indicative of the possibilities of growth of the muscles

I was in doubt at first as to what conclusions should be drawn from the results of the microscopic examination of the fragments of muscle removed in this case of congenital torticollis. The lesions were clearly those which have been described by Erb as existing in progressive muscular dystrophy, but they have been observed rarely in other conditions. I recalled the statement of Dr. Keen that deformity of the boy's legs had been noted and that there was probably more of interest in the case than the torticollis. I requested permission of Dr. Keen to see this boy, and he most kindly sent him to me. I found that the intense muscular atrophy, of which Dr. Keen had spoken, was probably progressive muscular dystrophy of the rather rare infantile form, and that my impression of the nature of the affection, formed by microscopical study of fragments of the sternocleidomastoid muscle and Dr. Keen's statements, was correct. There was no pseudo-hypertrophy in the case. By Dr. Keen's permission I shall report the clinical features later more in detail.[6] The point I should like to emphasize is that a diagnosis of this disease could be made with some probability from the microscopical examination of a section of one muscle.

It would be unjustifiable to regard these findings as pathognomonic of primary muscular atrophy; indeed, Erb himself by no means excludes all spinal involvement in the so-called idiopathic muscular atrophy, and is inclined to believe that where organic spinal changes are not detected, functional ones may possibly be the cause of this muscular wasting. The investigations of Müller, Schultze, and especially of Dejerine, have shown that hypertrophy of individual muscular fibres cannot be regarded as pathognomonic of idiopathic muscular atrophy, although at one time this view was quite generally held, and the studies of Hitzig, already mentioned, in which all of Erb's findings in muscular distrophy are described by Hitzig, as existing in infantile spinal paralysis, would warn us against making a similar mistake.

The existence of these remarkable findings in the muscle from a case of congenital torticollis, or more truly speaking, in congenital arrest in the growth of, and atrophy of muscles, does not justify us in assuming that the nervous system is not involved in such arrest. It is true that they are not seen after experimental section of nerve fibres, or after total destruction of the spinal trophic centres. In these cases hypertrophy of the fibres, vacuolation, segmentation, etc., are absent (Erb).

Destruction of certain nerve cells in the anterior horns of the cord, in anterior poliomyelitis, may lead to alterations of nutrition in the muscles, and secondarily to the production of the muscular changes described (Hitzig. Erb), and partial congenital arrest in the development of the spinal cord could in this way be the cause of the condition of the muscles in congenital or early infantile progressive muscular dystrophy.

(1) Erb, Deutsche Zeitschrift für Nervenheilkunde, Vol. I. (2) Koelliker, Handbuch der Gewebelehre, Vol. I, p. 140. (3) Golgi, quoted by Morpurgo and Bindi. (4) Dejerine and Sottas, Revue de Médecine, 1895. (5) Morpurgo and Bindi, Virchow's Archiv, Vol. 151, No. 2. (6) Spiller, Medical Record, July 2, 1898.

Alterations in the Spinal Cord in Azoturia

[FROM THE WILLIAM PEPPER CLINICAL LABORATORY]

JOSEPH SAILER, M. D.

I have recently, through the courtesy of the Veterinary Department of the University of Pennsylvania, been given the opportunity of studying the spinal cord removed from a horse that died of a disease known as *azoturia*. This is a peculiar disease occurring only in horses, particularly heavy, plethoric draft horses. It seems to be the result of over-feeding, especially with oats; and almost invariably develops if the horse, after standing a few days in the stable, with full diet, is suddenly obliged to do heavy work. The general pathology consists in cloudy swelling of the muscles, breaking down of the red blood corpuscles, hæmoglobinemia, hæmoglobinuria, fatty degeneration of the liver, and splenization of the marrow. The kidneys are at first normal, but in the course of a few days undergo parenchymatous degeneration. The symptoms are briefly as follows:—shortly after being taken out of the stable the horse becomes excited, then unsteady, and finally the hind legs become paralyzed and the horse falls to the ground. Anæsthesia apparently exists also, for pricking the skin causes no pain. The muscles are hard and tense; there is no fever; the appetite is not affected. There is usually retention of urine, which when drawn is coffee-colored, and in the course of about four days death occurs. Occasionally the front legs are also affected. Aside from the preliminary excitement, which may be ascribed to pain, there are no symptoms of cerebral involvement. The spinal cord, upon external examination, appeared perfectly normal, was of good consistency, and the membranes were not injected. Upon making section, a few dark spots were found scattered in the gray matter, usually around the blood-vessels, but no other changes were observed A few pieces were hardened in alcohol, and the rest in 10 per cent formalin solution. Upon staining sections by Nissl's method, extensive degenerations of the gray cells were observed. These practically exhibited every type that I have hitherto found described. The predominating alteration was a chromatolysis, with at the same time some shrinking of the cell. It stained diffusely, the nucleus was often obscure, or absent, the nucleolus was usually normal, but frequently surrounded by an irregular mass of chromatin. The protoplasmic processes were usually reduced to slight irregular projections, although this was not always the case, and sometimes the chromatin spindles were more distinct in them than in the protoplasm of the cell proper. These cells exhibited all degrees of intensity of staining, varying from a dark blue to a pale purple color. Vacuolization was less frequent, the vacuoles were usually situated at the periphery, were somewhat oval in shape, with the long axis parallel to the side of the cell, and rarely more than one was found in a single cell. An appearance of vacuolization was sometimes produced when the achromatic commencement of the neuroaxon was included in the section; and another change, which has recently been described by Nageotte and Ettlinger (*Presse Medicale*, 1898) was also found in some instances, that is the appearance of little clefts in the protoplasm of the cell substance. Shrinking of the cells was also observed in a number of cases; they became excessively small, but did not stain with unusual intensity. The nucleus was frequently placed eccentrically. The chromatin often stained as a dark irregular mass. In sections stained with hæmotoxylin, essentially the same appearance was produced. The chromophilic bodies were quite distinct. The changes in the nucleus were even more pronounced. Essentially the same appearances were produced by other methods, and no additional features were observed except that in sections stained with carmin it could be seen that the medullated fibres of the white matter were apparently entirely normal, and there was no proliferation of neuroglia in any part of the cord. The central canal was, as is normally the case in horses, lined by columnar epithelium, which, in frozen sections mounted in glycerine, could be seen to be ciliated. There was a slight gliosis outside of this epithelial layer. In no part of the cord, in any of the sections cut, were there any traces of round cell infiltration. The dark spots, to which I have already alluded, appeared to have been due to the staining of the tissue around a vein, probably by altered hæmoglobin. The frozen sections were usually stained by placing them in very dilute solutions of hæmatoxylin, or of the basic aniline dyes, and then were moulded in glycerine. It is perhaps worthy of note, that by this method, none of the cells appeared to contain chromophilic bodies, but consisted, apparently, of a homogeneous or faintly granular protoplasm that took the stain quite distinctly. The nucleus was enclosed in a distinct membrane, and there was also a distinct and deeply staining, but rather unequally thick membrane about the nucleolus, in which none of the so-called vacuoles could be found. In the more degenerated cells, of course, the nucleus could not be observed. These changes point very strongly, I think, to the presence of some toxic substance in the body. It is of course impossible to say what the nature of this toxic substance is, but from the history of these cases it seems not unlikely that some autointoxication from the intestinal tract occurs, that is probably not the result of inflammatory action.

Case of Rupture of the Aorta (without Aneurysm or Atheroma), with Evidences of Former Mesenteric Infarction.

FREDERICK A. PACKARD, M. D.

The following case is reported because of the comparative rarity of rupture of the non-aneurysmal aorta, and also because of the peculiar mesenteric lesion:

Robert G., white, *aet.* 75 years, was a tailor by occupation until his admission to the " out-wards " of the Philadelphia Hospital. He was there given employment in the tailoring department. While sewing he suddenly fell over to the floor in a "faint." He was at once brought over to Ward III. and still had his needle in his hand upon admission. He said that for three weeks he had had pains in his abdomen, but these were not marked until suddenly he had an attack of severe agonizing pain in the epigastrium just before he fell unconscious from his seat. He had been sitting quietly at his work, and had made no exertion for some time before the onset of pain. On admission to the hospital ward he was of an ashy hue, the lips devoid of color; the skin was cold and moist, his temperature 95° F. The pulse was almost imperceptible, but the heart beat only sixty-six times to the minute. There was much subjective dyspnea, the respiration being twenty-eight per minute.

I saw him shortly after his admission to the ward, and examined him as carefully as was permissible in his condition of shock. He was a spare old man, with pinched features, evidently suffering severe abdominal pain, which caused him to moan constantly and frequently change his position. The pupils were equal and reacted normally. The tongue was fairly clean and gave no evidence of digestive disturbance. The radials were decidedly calcareous. Careful examination of the thorax showed no sign of aneurysm, and the lungs were found to be normal except for emphysema. The apex of the heart was in the fifth interspace, 1.5 c. m. within the left nipple line. At the apex there was a systolic murmur which was well transmitted into the axilla. At the aortic cartilage there was a somewhat harsh systolic murmur, which was transmitted into the great vessels of the neck. At the pulmonary cartilage a double murmur was heard. The hepatic dullness extended from the fifth intercostal space to the costal margin. The splenic area was normal. The abdomen was rather flat, and there was an area of distinct resistance in the epigastric region slightly to the right of the median line. Over this area there was tenderness on pressure. In other respects the abdomen showed nothing. There was no hernia. The lymphatic glands in the left axilla were distinctly enlarged, but no other groups showed any abnormality. The urine was high-colored, had a specific gravity of 1025, and contained a small amount of albumin and granular and hyaline casts.

Physical examination, therefore, showed little to account for his manifestly grave condition. The first thought was, of course, rupture of an abdominal aneurysm. Careful search gave no evidence of the existence of such a lesion. Acute intestinal obstruction was considered and thought possible, but not probable. In view of the sudden onset, of the presence of an aortic obstruction murmur, and of marked atheroma of the peripheral arteries, and of intense abdominal pain, it was thought probable that the condition was produced by embolism of a mesenteric artery, such as had been recently reported by several physicians and surgeons in Boston. (It is to be pointed out that *recent* embolism of the mesenteric artery was thought of, and that the condition of the mesentery found at autopsy was not all suspected.)

The patient never rallied and died at midnight, about twelve hours after the onset of his acute illness. The autopsy was made at the patient's home, under very trying circumstances, by the resident physician, Dr. Raymond Spear. The following notes were made by him: Body of an old man, with extreme pallor of skin and mucous membranes. Rigor mortis fairly well marked. The abdominal cavity contained some blood-stained fluid. Both pleural cavities contained about a pint of blood-stained fluid. The layers of the pleural sac at the root of the left lung and the surrounding tissues were infiltrated with blood. The pericardium was filled with large clots of blood. The heart-muscle was flabby and had evidently undergone fatty degeneration. In both the aortic and mitral leaflets there were calcareous deposits. The lungs were emphysematous, and the base and root of the left lung were infiltrated with blood. The liver and spleen were normal. The kidneys showed marked interstitial change. Their capsules were adherent, and when removed left a granular surface. There was marked increase of interstitial connective tissue. In the much narrowed and shrunken cortex there were several small cysts. The pancreas was normal. The intestine was examined with care, owing to the previous suspicion of mesenteric embolism. For a distance of two feet upward from the ileo-cecal valve the ileum was at several points dark and apparently almost gangrenous. In the mesentery at these points were several areas of cicatricial appearance with considerable puckering. The aorta was very soft and flabby. The ascending portion of the arch was somewhat dilated. About two inches above the aortic valve leaflets there was a longitudinal crossed by a transverse tear. The outer coat was separated from the inner coats for some distance and was filled with recently clotted blood.

The heart and aorta and the diseased portion of mesentery were removed for further study. The aorta was very flabby and could readily be torn at any point by the use of but little force. Separation of the layers could readily be produced. The tear running transversely measured 1.5 c. m. in length, with slightly irregular edges, while the longitudinal tear looked more as though it might have been an artefact, and doubtless was made in removing or exposing the arch. The tear extended through all three coats and into the periaortic portion of the pericardial sac at the same level. There was no evidence of dissecting aneurysm although there was some dissection of the coats on all sides of the tear. This separation by escaped blood had every appearance of its having been recently formed just before death and corresponded in the extent of infiltration by blood with the general infiltration of all of the surrounding tissues.

Careful examination of the lower portion of the ileum and its corresponding mesentery showed a truly remarkable condition. The intestine was of a somewhat purplish hue, but did not seem softened at any point and, save for the discoloration, appeared to be unaltered. The mesentery near its attachment to the intestine was puckered in two large areas. Just above both of these areas the large arterial trunks running down in the mesentery were united by large, apparently compensatorily dilated vessels forming abnormally wide anastamoses. In the areas of apparently cicatricial tissue and in the space between them and the intestine there were no vessels visible to the naked eye. Careful examination of the mesenteric vessels by palpation and slitting showed no gross plug.

Pieces of the aortic wall were hardened and given to Dr. J. Dutton Steele, who very kindly made sections for me. All of the coats of the vessel showed advanced diffuse fatty degeneration.

The case was evidently an instance of spontaneous rupture of the aorta. The rupture occurred completely at one time, as was shown by the post-mortem appearances and also as was rendered likely from the clinical history, no second exacerbation in the symptoms having been noted, such as are usually present when the coats give way at different times. The immediate cause of death is difficult to determine. From the manner of death it might have been due either to hemorrhage *per se*, or to embarrassment of the heart's action from pericardial overfilling. While, as has been said, the provisional diagnosis of acute mesenteric embolism was made during life, this was shown at the autopsy to have been an error. By a curious coincidence, however, what to my mind is clear evidence of such an accident having previously occurred was found at autopsy. When this occurred can not be decided, but it evidently was at least many weeks before death. Source for such an embolus was found in the aortic obstructive lesion and in the highly atheromatous condition of the descending thoracic and abdominal aorta. No explanation other than a former mesenteric embolism could explain the unique appearance of the mesentery. It is hardly likely that the peculiar scar-like areas in the mesentery could have resulted from former breaking down of a mesenteric gland in view of the fact that there was no affection of other glands within the abdomen.

The reason for the mistake in diagnosis was the presence of intense epigastric pain with tenderness in the epigastric region. On looking up the literature of aortic rupture I find that this symptom is present in a large number of the reported cases, being especially remarked upon by Samuel Gordon (*The Medical Press and Circular*, March 25, 1874, p. 243), in his article entitled "Remarkable Case of Rupture of Aorta, in which epigastric pain was the principal symptom."

According to all authors, the male sex is, as would be expected, by far the more liable to rupture of the (not aneurysmal) aorta. Thus Broca's 29 cases comprised 19 males and 9 females; Chauvel's 18 cases comprised 10 males and 2 females; Martin-Dürr's 41 cases comprised 27 males and 14 females; Ames' and Townsend's 50 cases comprised 34 males and 14 females. The age at which rupture occurs is given by all authors as usually between 30 and 60, yet Sparks records a case occurring at 16 years and Martin Dürr one beyond 80 years.

The immediate cause is often unascertainable, as in the case herewith reported. This was true in 6 of Broca's 29 cases, in 7 of Chauvel's 18 cases, in 21 of Martin-Dürr's 41 cases, in 34 of Ames' and Townsend's 50 cases. From this apparently undiscoverable cause the exciting action ascends in severity from eating too hearty a meal up to the effort of stopping a runaway horse. Garland states that he has found the record of one case occurring during parturition.

The anatomical condition of the aortic wall is probably in no case normal, but in quite a large number of cases it is stated to have been healthy. In most of the cases that I have looked up where this statement is made, there is quite manifestly imperfect study. Broca found three statements that the aorta was healthy, 14 records where the intima and media were altered, and in 12 of his cases no statement as to the aortic walls is made. In 12 of Chauvel's 18 cases the walls were noted as healthy in 3; as altered, in 9 (6 of which showed atheroma). Martin-Dürr found the conditions described as healthy in 1, with dilatation in 13, atheroma present in 19, while there was hypertrophy of the left ventricle in 13. Ames and Townsend found the wall described as follows: In 4 (8 per cent) no atheroma; in 35 (70 per cent) atheroma, of which 8 showed ulceration, in 1 fatty, in 10 the condition not stated, in 1 a perforated tracheal ulcer, and in 1 a perforated esophageal ulcer. In 11 (22 per cent) there was more or less dilatation of the aorta, in 8 (16 per cent) it was stated to be not dilated. In 22 (44 per cent) of their cases the condition of the heart was not stated; in 17 (34 per cent) it was hypertrophied, in 3 of which dilatation was also present, while in 10 (20 per cent) the heart was healthy, in 5 (10 per cent) there being valvular disease. In 14 cases collected by Crisp the aorta was said to be healthy in 3. The intrapericardial portion of the aorta is by far the part most frequently ruptured. Thus in 28 of Broca's cases it was in this portion in 19, in other portions of the thoracic aorta in 7, in the abdominal aorta in 2. In 12 of Chauvel's cases it is given as in the pericardial portion in 10, in the rest of the thoracic aorta in 1, in the abdominal aorta in 1. In Martin-Dürr's cases the seat was described in 28, of which 15 were a little above the aortic valves, 5 in the ascending portion of the arch, 2 in the horizontal portion, and 6 at the junction of the ascending and horizontal portions of the arch. Of Ames' and Townsend's cases 50 per cent occurred within the pericardial portion, 10 (20 per cent) in the arch.

Death occurs at varying times after the rupture, depending upon the size of the rupture somewhat, but also to a great extent upon the completion of the rupture at one stroke or at two separate times (*rupture en deux temps*, Broca). The time elapsing between the first sign of rupture and death varies from a single moment to 23 days (Gordon's case). In Broca's group of cases 16 died suddenly, 2 in 15 minutes, 3 in an hour, while 4 others lived 5 hours, 24 hours, 45 hours, and 14 days respectively. Of Chauvel's collection 8 died immediately, while one lived 15 days. Martin-Dürr divides his deaths into sudden (6), slow at one time (5), and slow in two times (12).

Broca made the distinction between death from hemorrhage *per se* and death from cardiac compression. Lesage explains death somewhat differently. He says that if hemorrhage into the pericardium be small (80 grams) death occurs from pressure upon and excitation of nerves, if large (500 to 800 grams or more) it is produced by compression of the heart.

Brouardel and Vibert have recorded a remarkable case of rupture of the aorta at two places.

There are two points presented by my case to which I would again draw attention. One of these is the intense epigastric pain which helped to cause an erroneous diagnosis to be made. This would appear to be a very common symptom in these cases, although I regret that in going over the literature of the subject I failed to note the relative frequency of its presence. Speaking from memory I should say that by far the larger part of the cases that I studied presented this as a marked symptom. The other point is the curious slowness of the pulse in spite of the feeble cardiac action and the presence of severe pain. This slow pulse-rate was also present to a marked degree in one of Chauvel's cases, and in a large number of reported cases the pulse is not accelerated to anything like the rate that would be expected in ordinary collapse, especially when this was associated with pain. It must, I presume, be attributed to pneumogastric stimulation by the effused blood, as it is hardly likely that there was sufficient pressure to paralyze the accelerator nerves of the heart.

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DISCUSSION.

DR. SAILER.—The mesenteric circulation may be viewed as a sort of safety-valve for the aorta, because the dilatability of the mesenteric vessels and their large capacity permit of a large overflow into them. It seems to me possible that if there was a mesenteric infarct in this case it may have predisposed to the aortic rupture.

Enlarged Cirrhotic Liver.

AUGUSTUS A. ESHNER, M. D.

A man, 37 years old, who had been employed as a postman, was visited at his home by one of the resident physicians of the Polyclinic Hospital on June 4, and found with a temperature of 102.4°, a pulse of 120 and respirations of 44. He was decidedly jaundiced, and was delirious. The skin was hot and dry. The abdomen was tender, greatly distended and tympanitic. The breath-sounds were clear and no cardiac lesion was detected. It was learned that the patient had been employed as a postman until April last, when he lost his position, and he had since been out of employment. In October, 1897, he began to drink heavily and he had continued so to do. The man was admitted to the Polyclinic Hospital in the evening of June 4. On physical examination the action of the heart was found to be deliberate and its sounds clear. On percussion there was impairment of resonance posteriorly on the left side, but the breath-sounds were transmitted; anteriorly no peculiarity was detected. The pulse was rapid and unsustained. In addition to the distention and tenderness of the abdomen, the superficial veins were found distended, especially upon the right side, but there was no suggestion of a caput Medusæ. The liver-dulness extended from the fifth interspace in the nipple-line to the costal margin and for 5 c. m. below the ensiform cartilage in the median line. No enlargement of the spleen was detected. The urine was of amber color and clear, with a specific gravity of 1012, of neutral reaction, free from albumin and sugar, reacting to tests for chlorids and containing considerable biliary coloring-matter. The temperature ranged between 102.4° and 100°; the respirations between 32 and 52, and the pulse between 84 and 152. Consciousness did not return during the five days that the patient was under observation, and from the second day the urine and feces were passed involuntarily. Subcutaneous infusion of a pint of normal saline solution was practised on the second, third and fourth days, and was in each instance followed by a reduction in temperature of from 1° to 1.2° in the course of an hour and a half. An examination of the blood was made, but it failed to cause agglutination of typhoid-bacilli.

Upon post-mortem examination, 120 cu. c. m. of serous fluid were found in the abdominal cavity. The liver was enlarged, although it did not extend notably beyond the costal margin. The transverse fissure was situated exactly in the median line, the left lobe projecting considerably to the left. The organ was granular and mottled on its surface, and on section it presented a nutmeg appearance. Its structure was quite dense. It weighed 3000 grams. On its under surface, beneath the capsule, were several dilated veins. The gall-bladder contained a considerable amount of limpid, aqueous fluid. The ducts were patulous. The spleen was enlarged, weighing 260 grams; there was a small supernumerary spleen. The stomach was the seat of an active, acute inflammation. The kidneys were enlarged and in a state of chronic parenchymatous inflammation. The right weighed 240, the left 260 grams. The pericardium contained between 15 and 20 cu. c. m. of a blood-stained serous fluid. The right side of the heart was flabby and relaxed and the auriculo-ventricular orifice was dilated. The left ventricle was partially contracted. The mitral orifice was of normal size and the leaflets of the valve were thickened, but soft. The pulmonary artery contained a few spots of atheroma, while the aorta was much more markedly diseased. The right lung was slightly adherent at the apex, where there was a partially fibroid and partially softened area that tore on removal.

The left lung was bound tightly to the chest wall by the thickened pleura, each of the two layers of which were about half a centimeter thick. At the apex of the lung was a small, partially fibroid, partially calcareous area.

In the massive enlargement of the liver, in the enlargement of the spleen, in the presence of deep jaundice, of delirium, of elevation of temperature, in the absence, relatively, of ascites, of notable enlargement of the abdominal veins, of hemorrhages, the conditions in this case conform with those of hypertrophic or biliary cirrhosis. The enlarged liver was, however, granular on its surface and free from greenish-yellowish discoloration on section (perhaps because the jaundice had not been of long duration). The history of alcoholism is scarcely of differential etiological weight.

June 9, 1898.

Dilatation of the Heart—Atheroma of the Aorta—Pulmonary Infarction.

AUGUSTUS A. ESHNER, M. D.

The specimens that I have to present were derived from the body of a colored man, 33 years old, a teamster by occupation, who was admitted to the Philadelphia Hospital, under my care, June 3, 1898, in a condition of such marked general anasarca as I have never seen, and complaining especially of dyspnœa and palpitation of the heart. He had also at times taken alcohol to excess, but he denied syphilitic infection. There was a history of heavy lifting, with consequent strain, a year and a half before. The man dated his symptoms back three months. They began with pain in the lumbar region, which was shortly followed by shortness of breath on exertion. Later dyspnœa became constant and it had been so for six weeks. The lower extremities began to swell early and they continued swollen throughout. There was at times slight vertigo, but not sufficient to cause the patient to fall. The man was large and well nourished. He presented a distressed appearance and lay with his mouth open, breathing with great difficulty. Respiration was rapid, shallow and diaphragmatic. The vessels of the neck exhibited visible pulsation. The apex-beat of the heart could not be seen, although it was felt in the fifth intercostal space in the left mammillary line. The area of cardiac dulness extended horizontally from a point an inch to the right of the right border of the sternum to the left mammillary line and vertically from the second to the fifth interspace. On auscultation the first sound of the heart was replaced by a blowing systolic murmur transmitted to the axilla. At the aortic cartilage also a soft, blowing, systolic murmur was heard, transmitted to the vessels of the neck, together with a harsh diastolic murmur. A systolic murmur was heard also in the pulmonic area. The pulse was strong, but not sustained. The pulmonary percussion-resonance was impaired anteriorly on both sides. The breathing was roughened and occasional moist rales could be heard. The respirations ranged between 52 and 60, the pulse between 96 and 128. The urine contained albumin and hyaline and granular tube-casts. The condition of the patient grew progressively worse and death ensued about forty-six hours after his admission to the hospital.

Upon post-mortem examination the abdominal and pericardial cavities were found to contain a moderate and the pleural cavities a more considerable amount of serous fluid. The antero-lateral aspect of the right lung presented a circumscribed area that was the seat of a recent fibrinous deposit, beneath which were found two large infarcts, one of which was in process of softening. The lower lobe of the left lung also contained a large infarct. Throughout both lungs were numerous venous thrombi, varying in size from a millet-seed to a lentil. The lungs were œdematous. The right weighed 650, the left 620 grams. The heart was greatly enlarged in all of its dimensions and its several cavities were dilated; it weighed 740 grams. Beneath the epicardium in many places were numerous minute linear extravasations of blood. Between the layers of the left posterior leaflet of the pulmonary valve was an extravasation of blood. The pulmonary artery presented several small dots of atheromatous change. The right auriculo-ventricular orifice was much dilated; the left was of normal caliber. The tricuspid leaflets exhibited no morbid alteration; those of the mitral were slightly thickened. The aorta, including the valve-leaflets, was the seat of extensive and apparently active atheromatous change and it was partly suffused with blood. The spleen was small and fibroid; it weighed 90 grams. The kidneys were large, firm and exhibited the changes of a chronic parenchymatous inflammatory process; the right weighed 320, the left 290 grams. The suprarenal bodies were hard, the cortex and medulla were readily differentiable. The gall-bladder was enlarged and elongated. It contained a couple of drams of mahogany-colored bile, and its mucous membrane, which presented a honeycombed appearance, was deeply stained of the same hue. The liver seemed to be undersized and, on section, it presented a nutmeg appearance; it weighed 1750 grams.

The extensive atheroma of the aorta, with beginning degenerative changes of like character in the pulmonary artery, was a marked feature of this case, particularly on account of the relative youth of the patient. The denial of infection on his part does not, of course, exclude the possibility of syphilis. Besides, there was an admission of alcoholic excess; and it is not impossible that the laborious work of the patient in the lifting of heavy weights as a teamster may have had something to do with aggravating if not inducing the aortic disease.

June 9, 1898.

Pseudo-Parasites in the Stools of a Child.

FREDERICK A. PACKARD, M. D.

The specimens presented were brought to me by Dr. A. W. Myers, who told me that they were passed by a child aged two years, living in a town in West Virginia. The child was fed upon a general mixed diet. About a month ago she began to complain of pain on defecation, and it was noticed that her abdomen was swollen and tympanitic. On examination of the stools, these foreign bodies were found to be present in large numbers, and they have been present in every movement since. The stools are passed with considerable pain, straining and tenesmus. At first the movements contained considerable mucus, but lately blood has also been observed. A fifteen months' old child, living in the same town, is said to have passed similar bodies, with corresponding symptoms, to have developed convulsions and died. In the latter child the diet had been breast or cow's milk exclusively.

The material brought to me consists of dark-brown thread-like bodies, measuring from 0.5 c. m. to 1.5 c. m. in length and from 0.5

to 1 m. m. in thickness. They have a fairly uniform structure, and to the naked eye show that they are composed of three layers—two rows of bead-like, minute, dark-brown bodies arranged in parallel lines, enclosing a central clear layer. On magnification they are found to be of vegetable structure, the dark rows being composed of large oval cells containing chlorophyl placed end to end, the clear layer containing loose fibrillar material and a central spiral vessel. The general arrangement much resembles sections of leaf, but it is difficult to explain the manner in which such cross-sections could have been produced. Further details regarding diet might throw some light upon the source of the material. Without positive knowledge regarding the diet of the child, it seems to me that spinach would be the most likely source for such bodies. I have never seen vegetable fibre of exactly this arrangement in the examination of quite a large number of stools, but shall take advantage of my earliest opportunity to investigate the stool after the eating of spinach. The pain at the time of voiding the material may depend upon some factor other than this material.

It would seem that the supposed resemblance between this case and the milk-fed baby must have been erroneous, as no purely milk-fed child could pass material of this nature. Neither of the cases has been seen by Dr. Myers, and our statements regarding them are from non-medical sources.

June 23, 1898.

Chronic Interstitial Nephritis (Cystic Kidneys): Hypertrophy of the Heart: Hemorrhage into the Pons Varolii.

AUGUSTUS A. ESHNER, M. D.

A colored man, 70 years old, who had been a sailor most of his life, was admitted to the Philadelphia Hospital under my care with symptoms of uremia. He admitted alcoholic excess, but denied venereal infection. The area of cardiac dullness was increased and the second sound of the heart accentuated; the urine contained albumin and tube-casts; but there was no anasarca. Muscular twitching and delirium set in, control of the sphincter of the bladder was lost, sopor passed gradually into coma and death resulted amid symptoms of respiratory failure. The temperature rose only a fraction of a degree above 99°, and for the last few days of life it was subnormal, falling as low as 95°. Respirations declined to 13 and the pulse to 64. Nystagmus was noted at times, but trismus was not observed. Upon post-mortem examination the lungs were found edematous and emphysematous. The heart was enlarged and hypertrophied, weighing 460 grams. Its valves were competent, its orifices unobstructed. The aorta was advancedly atheromatous. The kidneys were small, cystic and granular; the right weighed 155, the left 120 grams. The vessels at the base of the brain were thickened; those in the fissures were atheromatous. The meninges of the convexity were somewhat opaque. In the left half of the pons, midway between the median raphe and the superficial origin of the fifth nerve, was a dark-red, apparently recent extravasation of blood about 2 mm. in its several diameters. Some 4 mm. further forward, almost in the middle line and at a depth of 7 mm. from the surface, was a second extravasation of similar appearance and about the same transverse and vertical measurement, but somewhat longer. The ependyma of the lateral ventricles was rough and granular. *June 23, 1898.*

Cancer of the Head of the Pancreas.

W. B. JAMISON, M. D.

T. I., male, *æt.* 78, had a family and personal history of no special interest. For some months before his death he complained of pain in the abdomen and became jaundiced, which deepened progressively. Emaciation progressed, his digestion was poor and associated with attacks of alternating diarrhœa and constipation. For some weeks the abdomen was swollen, due to peritoneal effusion, and hemorrhoids of long standing became very large and painful.

The autopsy was made sixteen hours after death. Jaundice was extreme; the abdomen distended. The peritoneal effusion consisted of about a gallon of deeply yellow fluid. The thoracic organs showed only senile changes. The kidneys showed not only granular changes, but also marked fatty degeneration of the cortex. There was a moderate increase in the connective tissue of the liver. The gall-bladder was enormously dilated. The bile ducts passed through a hard mass in the head of the pancreas. The mass was about an inch and a half in diameter, hard, and on section of glandular appearance. Microscopical examination confirmed the diagnosis of carcinoma. *June 23, 1898.*

Intestinal Obstruction by a Peritonitic Band.

DAVID RIESMAN, M. D.

The patient from whose body this specimen was obtained at autopsy was an old man, seventy-two years of age, who had been a denizen of the Blockley out-wards until his admission to the Philadelphia Hospital two days ago. All that is known of his history—and for these data I am indebted to the kindness of Dr. Halloway, of the resident staff—is that on Sunday, May 8, he had been seized with sharp pain in the abdomen, for which he received some calomel. He had a small bowel movement. During the night he was awakened by severe abdominal pain and vomiting. On the following day efforts were made to secure free purgation, but the patient had only a slight movement, and rapidly grew weaker. On his admission to the hospital, May 10, 1898, there was tympany over the left side and diminished resonance over the right side of the abdomen, together with some distension and tenderness. The last was especially marked about midway between the umbilicus and the right anterior superior spine of the ileum. Manipulation of the abdomen brought on stercoraceous vomiting.

A laparotomy was promptly performed to relieve the intestinal obstruction so clearly indicated by the symptoms, but the patient died before the completion of the operation.

At the autopsy a loop of ileum was found strangulated under a band of adhesion, the remains of an old peritoneal inflammation. This band passed from the side of the large bowel, near the junction of the cecum and ascending colon, over to the lateral wall of the abdomen, and measured 2 c. m. in width at its intestinal and 6½ c. m. at its parietal end, spreading out here into a broad base. The band bridged a foramen which admitted two fingers and was formed by the under surface of the adhesion and another narrow band passing downward from its under surface. Through the orifice thus produced, which had a sharp edge, a loop of ileum 10½ c. m. in length had slipped and had become strangulated. The loop was deeply congested and dark red in color, but not gangrenous. The other parts of the intestine were distended with gas and liquid, and there was a little bloody fluid in the peritoneal cavity.

Interesting, and perhaps of some influence in hastening the man's death, are the kidneys, which I here exhibit. They are, as you see, very asymmetric, the right being only about one-third the size of the left—the latter weighs 180, the former 60 grams. Both are deeply scarred and puckered and are the seat of an advanced cirrhosis.

The heart, as would be expected, was hypertrophied.

May 12, 1898.

Cystic Kidneys.

J. D. STEELE, M. D., for J. P. CROZER GRIFFITH, M. D.

The specimens were obtained from an adult male who had been suffering from symptoms resembling uremia. They consist of two very large cystic kidneys, each 18 c. m. in length. The cysts are grouped in masses resembling bunches of grapes, as is usually the case in this type of cystic kidney. While the development of such extreme cystic degeneration must have been of comparatively short duration, it is generally considered that the tendency to such a change is some error in development.

DISCUSSION.

DR. ARNOLD recalled a case in a person of sixty years; the urine was practically normal.

DR. SCHREINER had seen a case in which the liver presented an analagous condition.

DR. WESTCOTT recalled two cases which occurred in brother and sister; one died of uraemia.

DR. RIESMAN.—That cystic change in the kidneys, of this character, may be congenital, is demonstrated by the fact that it sometimes causes a hindrance to labor. Regarding the pathogenesis of the condition, one theory is that it is due to obstruction of the papillæ, another that it represents an adenomatous hyperplasia. It is nearly always bilateral. Clinically it is of interest to note that despite the presence of so many cysts the tumors usually do not give fluctuation, a point of diagnostic value. Last June I showed a pair of kidneys quite similar to these, but of a much lighter color, from the museum of the Philadelphia Hospital.

May 12, 1898.

Calculus of the Spleen.

JOSEPH SAILER, M.D.

The following specimen was removed from an old man, and is only interesting on account of the discovery, in the substance of the spleen, of a small round calculus, about one-half c. m. in diameter, with a smooth surface. Originally it was white, but it appears to have become stained in the hardening solution. It probably represents a calcified thrombus. According to Orth, these calculi are not uncommon in the spleen, but I have failed to find reference to them in other text-books. They are probably a result of varicosities in the splenic veins in which the thrombi are formed.

Typhoid Ulcer in the Appendix.

JOSEPH SAILER, M.D.

The patient, a negro girl, 19 years of age, came to Philadelphia from Virginia four weeks ago. A few days before her admission to the hospital, she became ill and a diagnosis of typhoid fever was made. Upon her admission, the Widal action was obtained, but was not entirely characteristic. The following day she had a severe convulsion which was repeated a number of times and was supposed to be uremic. Death occurred the same evening. At the autopsy, typical typhoid ulcers were found in the ileum, and the kidneys were in a state of intense cloudy swelling. The appendix was about 15 c. m. long and greatly distended. The serous coat was injected, but there was no lymph upon it and it was not adherent. Upon opening it a large oval ulcer was found near its junction with the caecum, and nearer the tip, the mucous membrane was somewhat swollen and contained a few minute ulcers. The condition is rare, and was so pronounced in this case, probably, on account of the unusual size of the appendix. *May 26, 1898.*

Minute Hemorrhage into the Pons.

F. A. PACKARD, M. D.

A solitary, minute, cherry-red hemorrhage lies in the left side of the pons, sharply circumscribed, and is 2 m. m. in diameter. No symptoms attributable to the hemorrhage were noted during life. The specimen was removed from the body of a man dying of typical chronic interstitial nephritis, with hypertrophy of the heart and intense atheroma of the circle of Willis.

Multiple Abscesses of the Liver Secondary to Chronic Purulent Peritonitis.

F. A. PACKARD, M. D.

The specimen presented a most extraordinary honey-combing of the liver by abscesses apparently following the portal distribution. An abscess was also present in the wall of the gall-bladder.

June 23, 1898.

Colloid Cancer of the Stomach.

J. P. ARNOLD, M. D.

The specimen herewith presented shows a large colloid cancer involving almost all of the posterior wall and greater curvature of the stomach, extending from the cardiac orifice almost to the pylorus, though not causing obstruction. The duodenum is bent upon itself about two inches from the pylorus and is held in this position by adhesions in such a manner as to prevent a free flow of its contents onward. The growth in the stomach is diffuse in character and of a gelatinous consistence. It projects from one-half to one inch above the surface of the mucous membrane. The peritoneal surface is not involved. None of the other organs were affected.

The points, of interest in the clinical history of the case, are as follows: There were no marked symptoms observed until about four months ago, when the woman began to complain of flatulence, variable appetite, and diarrhœa. After that time she had steadily lost weight. About two weeks ago vomiting became quite frequent. The vomited material was of a dark brown color. She never vomited any blood. At the time the vomiting became severe she first noticed a lump in the region of the umbilicus.

Physical examination made six days before death, when the patient was first seen, revealed the following condition: Walls of abdomen rigid, marked with striæ. The lower epigastric region is quite prominent and is tympanitic. No distinct mass can be outlined. The prominence in the epigastrium has the characters of a dilated stomach. There is very marked tenderness over this area. Examination otherwise negative.

At the autopsy it was found that the cancerous mass was so situated behind the left costal margin and covered by the liver that it could not have been palpated. The prominence in the epigastrium was the dilated pyloric end of the stomach.

It seems strange with such extensive involvement of the stomach that symptoms had only been noticed for four months before death. The growth must either have been of very rapid development or had existed for some time with very little disturbance of digestive function.

Atrophy of the Kidney from Obstruction of the Ureter.

J. P. ARNOLD, M. D.

The specimen shows quite typically the changes which take place in the kidney from ureteral obstruction when there is no marked infection. The ureter in this case was obstructed about two inches from the bladder, and from this point onward existed only as a fibrous cord.

The kidney is small, the structure has almost entirely disappeared and all that remains is a mass of cysts, filled with a semisolid yellowish material, made up largely of uric acid, urates and degenerated epithelium.

The cause of obstruction could not be ascertained.

A Carcinomatous Polyp of the Cervix Uteri.

H. L. WILLIAMS, M. D.

Polypoid growths from the mucous membrane of the cervical canal and from the body of the uterus are of comparatively frequent occurrence, though a cervical origin is more common than that from the fundus. Pick makes the following convenient classification of polypi arising from the cervix. These growths are divided into: (1) Inflammatory polypi and (2) real new growths. Under inflammatory polyps are included all the slight hyperplastic inflammatory projections from the cervical mucous membrane. The microscopical appearance in these cases is identical with that found in chronic hyperplastic endocervicitis.

The real new growths may be separated into (*a*) benign and (*b*) malignant growths. Benign polypi may be again subdivided into the (1) fibromatous polypi, (2) the adenomatous polypi, and (3) adeno-fibromatous polypi. The malignant polypi may be either (1) carcinomatous or (2) sarcomatous. Although recognized and occasionally described in the literature, carcinomatous degeneration of cervical polypi is of such rarity that the following case from the Pathological Laboratory of the University of Pennsylvania is of interest. No similar case has before occurred in the University Hospital service, nor has such a case been found in the laboratory at Johns Hopkins University.

This patient was under the charge of Dr. B. C. Hirst in the University Hospital. The history of the case is as follows: Mrs. C., *aet*, 48 years, has been married twenty-four years. She has had no children and no miscarriages. Menstruation has always been regular, and has lasted from five to seven days. For the last three years the menstrual flow has lasted but two days and has been painful the entire time. Leucorrhœa has been profuse and recently of an exceedingly bad odor. The patient has complained of pain in the back, pain in both sides, and pain along the lines of Poupart's ligaments and across the abdomen. Micturition has been frequent, imperative and painful. The abdomen has been somewhat enlarged upon the left side. The bowels chronically constipated. The patient first noticed pain upon the left side three years ago, followed by painful menstruation and abdominal enlargement. At the examination upon admission to the hospital a large, soft, sloughing mass was found just inside the external os, and completely filling the cervical canal.

At the operation the polyp, about the size of a horse-chestnut, was found to be attached by a firm pedicle to the upper left wall of the cervical canal. The cervical canal was enormously dilated and the cervical walls only a few millimetres in thickness. The mucous membrane of the right side of the canal, against which the polyp rested, was ulcerated and sloughing.

The specimen consists of a soft, sloughing, irregular polypoid mass, measuring 1¼ in. in length, and 3 in. in circumference. The cut surface of the pedicle measures ⅝ in. by ½ in., and is white and healthy in appearance. Sections were made from the body of the growth, and from the surface of the pedicle. The preparations from all the lower portions and centre of the growth show large nests of epithelial cells, in a somewhat glandular arrangement, separated by narrow septa of fibrous tissue. The entire growth has been infiltrated with cell nests to within a quarter of an inch of the cut surface of the pedicle. No cervical glands can be found in the preparation. The section from the cut surface of the pedicle shows apparently healthy fibres and muscular tissue throughout, except at the edges of the specimen, where a few nests of epithelial cells can be distinctly seen. One small cellular nest of epithelial cells can also be seen at a considerable distance within the margin of healthy tissue, so that a prognosis of complete eradication of all malignant tissue must be guarded, as will be seen from the microscopic sections.

June 23, 1898.

INDEX.

ABBOTT, A. C., discussion, 33, 39, 56, 167
Abscess of liver, 233, 305
  of lung, 39
Adenoma, malignant, of intestine, 91
Amyloid bodies in central nervous system, 277
Anders, J. M., and Jos. McFarland, pneumothorax with discovered perforation, 56
Aneurysm of aorta, 102, 178, 183, 185
  dissecting, of aorta, 40, 184
  of heart, 21
Anomaly of basilar artery, 64
Aorta, aneurysm of, 102, 178, 183, 185
    dissecting, of, 40, 184
  atheroma of, 297
  rupture of, 241, 288
Appendix vermiformis, foreign body in, 55
  inflammation of, 55
    with secondary pylephlebitis, 233
  typhoid ulcer in, 304
Arnold, J. P., atrophy of kidney from obstruction of ureter, 306
  colloid cancer of stomach, 305
  discussion, 8, 38, 303
  gastric carcinoma, 40
  tumor of dura mater, 19
Atheroma of aorta, 297
Azoturia, alterations in spinal cord in, 286

BASILAR ARTERY, anomaly of, 64
Basophile cells, 64
Bicycle rider, heart and lungs from, 181
Bile-duct, common, carcinoma of, 145
    gall-stone in, 229
    obstruction of, 72
  cystic, obstruction of, 252
Blackburn, A. E., and E. L. Duer, colloid cyst of ovary, involving splenic capsule, 104
Blood in diseases of cardio-vascular system, 134
  films, box for carrying, 150
  vessels, diseases of, 126
Bowel, multiple outgrowths from serous surface of, 143
  strangulation, internal, of, 35, 302
Bright's disease, pericarditis in, 182

CÆCUM, carcinoma of, 246
Calculus of spleen, 304
Cancer (see Carcinoma).
Carbolic acid, poisoning by, 39
Carcinoma of bile-duct, common, 145
  of cæcum, 246
  of gall-bladder, 9
  of liver, 250, 260
  micro-organisms, so-called, of, 221
  of pancreas, 145, 301
  of peritoneum, 85, 221
  of rectum, 85, 250
  of stomach, 29, 40, 62, 305
Carcinomatous polyp of cervix uteri, 307
Cardio-vascular system, diseases of, 109
    blood in, 134
    urine in, 138
Cattell, H. W., carbolic acid poisoning, 39
  discussion, 64
Cerebral hemorrhage, 37
Chestnut, J. E., calf's eye with tuft of hair on cornea, 184
Cholangitis, suppurative, 72
Chorio-epithelioma, 79
Choroid plexus, pathology of, 273
  tumor resembling, 276
Cirrhosis of kidney, 105
  of liver, 69, 295
Coccidium oviforme, 12

Colloid bodies in central nervous system, 277
  cancer of stomach, 305
  cyst of ovary, 104
Color perception, pathology of, 161
Coplin, W. M. L., description of new apparatus, 148
  diffuse tuberculosis of testicle. 267
  discussion, 103, 142, 156, 225, 273
  primary cancer of liver, 260
Coronary artery, right, obliteration of orifice of, 243
Cyst, colloid of ovary, 104
  dermoid, 180
  echinococcus, of liver, 106
  of kidney, 300, 303
Cystic duct, obstruction of, 252

DA COSTA, J. C., discussion, 44
Daland, Judson, aneurysm of arch of aorta, causing death by rupturing into left bronchus, 185
  appendix vermiformis with pin in situ, producing interstitial appendicitis, 55
  discussion, 157
Deciduoma malignum, 79
Dermoid cyst, 180
Dissecting aneurysm of aorta, 40
Dougherty, S. W., discussion, 237
Duer, E. L., and A. E. Blackburn, colloid cyst of ovary, involving splenic capsule, 104
Dura mater, cranial, tumor of, 19
  spinal, tumor of, 41

ECHINOCOCCUS cyst of liver, 106
Edsall, D. L., discussion, 156, 249
  typhoid fever with irregular lesions, 157
  and Joseph Sailer, peculiar bodies found in central nervous system, 96
Embolus, hepatic, in lung, 184
Endocarditis, ulcerative, 245
Endocardium, diseases of, 122
Enteroptosis, congenital, 154
Epiphysitis, acute, complicating typhoid fever, 151
Eshner, A. A., case of uremia, 5
  chronic interstitial nephritis (cystic kidneys), hypertrophy of heart, hemorrhage into pons varolii, 300
Eshner, A. A., discussion, 8, 71, 79
  dilatation of heart, atheroma of aorta, pulmonary infarction, 297
  enlarged cirrhotic liver, 295
Extra-uterine pregnancy, 145, 168
Eye of calf, with tuft of hair on cornea, 184

FIBROMA, intracanalicular, of both mammary glands, 238
Fisher, H. M., aneurysm of aorta rupturing into pleural cavity, 184
  syphiloma of liver, 103
Fussell, M. H., mitral stenosis, 19

GALL-BLADDER, carcinoma, primary, of, 9
Gall-stone in common bile-duct, 229
Ganglion cells, nucleolar degeneration of, 217
Gangrene of lung, 63
  symmetrical, of extremities, 73
Garritee, C. J., discussion, 33
Gaylord, H. R., discussion, 172
  hepatic embolus in lung, 184
  illustrations of cardiac lesions, 184
Gittings, J. C., case of acute appendicitis, with secondary pylephlebitis and multiple abscesses of liver, 233
  discussion, 237
Granular bodies in central nervous system, 277
Griffith, J. P. C., and J. D. Steele, cystic kidneys, 303

HAMILL, S. Mc., aneurysm of arch of aorta, 102
  discussion, 103
Hand, Alfred, Jr., acute general miliary tuberculosis, 19
  tuberculosis of myocardium, 92
Hare, H. A., specimens of cardiac lesions, 226
  spleen and liver from case of leukæmia, 227
  and H. F. Harris, miliary carcinomatosis of peritoneum, with microscopic slides showing so-called micro-organisms of cancer, 221
  discussion, 217

Harris, H. F., two new methods of staining axis cylinders of nerves in fresh state. Some microchemic reactions of toluidin blue, 208
    and H. A. Hare, miliary carcinomatosis of peritoneum, with microscopic slides showing so-called micro-organisms of cancer, 221
Heart, aneurysm of, 21
    from bicycle rider, 181
    capillaries in muscle fibres of; absence of veins of smallest size from, 186
    dilatation of, 297
    diseases of, 109
    hypertrophy of, 300
    lesions of, Schmoerl's stereopticon views of, 184
        specimens of, 226
Hektoen, L., discussion, 141
Hernia, inguinal, congenital, 60
Herrick, J. B., discussion, 141
Hour-glass stomach, 183
Hughes, W. E., carcinoma of common bile-duct and of head of pancreas, 145
    discussion, 60, 71, 103, 144
    diseases of pericardium, 109
    heart and lungs from bicycle rider, 181
    pericarditis in Bright's disease, 182
    rupture of kidney and spleen, 181
    specimens from two cases of Weil's disease, 65
Hyaloid bodies in central nervous system, 277
Hydronephrosis, congenital, 253

ILEUM, outgrowths from serous surface of, 143
Infarction of lung, 297
    of mesentery, 288
Infundibulum, tumor of, 64
Inguinal hernia, congenital, 60
Intestine, adenoma, malignant, of, 91
    obstruction of, by band, 35, 302
Intracanalicular fibroma of mammary gland, 238

JAMESON, W. B., cancer of head of pancreas, 301
Jopson, J. H., discussion, 153

KEEN, W. W., and W. G. Spiller, two cases of wrist drop from injury of musculo-spiral nerve from fracture of humerus, 100
Kelly, A. O. J., multiple sarcomata, 105
Kidney, atrophy of, from obstruction of ureter, 306
    cirrhosis of, 105
    cysts of, 300, 303
    rupture of, 181
    tuberculosis of, 34
Koch's Tuberculin R., review of literature of, 49

LEUKÆMIA, 227
Liver, abscess of, 233, 305
    carcinoma of, 250, 260
    cirrhosis of, 69, 295
    echinococcus cyst of, 106
    in leukæmia, 227
    sarcoma, melanotic, of, 33
    syphiloma of, 103
Lung, abscess of, 39, 245
    gangrene of, 63
    hepatic embolus of, 184
    infarction of, 297
Lungs from bicycle rider, 181

MALIGNANT adenoma of intestine, 91
Mammary gland, intracanalicular fibroma of, 238
Mast cells in sarcoma, 183
McFarland, Joseph, case of aneurysm of aorta, with rupture into right pleural cavity, 178
    case of symmetrical gangrene of all four extremities without discoverable cause, 73
    coccidium oviforme, 12
    discussion, 60, 79
    and J. M. Anders, pneumothorax, with discovered perforation, 56
Meckel's diverticulum, 193
Meigs', A. V., demonstration in sections of human heart, of which arteries were injected, of presence of capillaries within muscle fibres; and of absence of veins of smallest size from heart, 186
    discussion, 142, 244
Melanotic sarcoma of liver, 33
Melkinow-Raswedenkow method of preserving organs, 63

Meninges, cranial, tumor of, 19
  spinal, tumor of, 2, 41
Mesentery, infarction of, 288
Mitral stenosis, 19, 226
Morgan, A. C., cancer of cæcum, 246
Morton, S. M., and A. F. Witmer, tumor of spinal meninges, 2
Musculo-spiral nerve, injury of, with wrist drop, 100
Musser, J. H., cerebral hemorrhage, 37
  discussion, 11, 143
  melanotic sarcoma of liver, 33
  President's address, 1
  and J. D. Steele, diseases of myocardium, 116
Myocarditis, fibroid, 183
Myocardium, diseases of, 116
  tuberculosis of, 92

NEPHRITIS, chronic, interstitial, 300
Nerves, axis cylinders of, new methods of staining, 208
Nervous system, central, amyloid, colloid, hyaloid and granular bodies in, 277
  changes in, after aseptic injury, 45
  nucleolar degeneration of ganglion cells of, 217
  peculiar bodies found in, 96

OMPHALOMESENTERIC duct, 193
Ostheimer, Maurice, gangrenous carcinoma of stomach, 62
  slides showing basophile cells, 64
Ovary, cyst of, colloid, 104

PACKARD, F. A., case of gall-stone in common duct, with intermittent fever, 229
  case of rupture of aorta (without aneurysm or atheroma), with evidences of former mesenteric infarction, 288
  discussion, 8, 19, 60, 145, 147, 237, 243
  diseases of blood-vessels, 126
  minute hemorrhage into pons, 305
  multiple abscesses of liver secondary to chronic purulent peritonitis, 305
  multiple outgrowths from serous surface of ileum, 143
  pseudo-parasites in stools of child, 299
Pancreas, carcinoma of head of, 145, 301
Paraffin oven, description of, new, 148
Parasites, pseudo-, in stools of child, 299
Pearce, F. Savary, case of intra-canalicular fibroma of both mammary glands, 238
Pepper, Wm., Jr., case of cancer of rectum and liver, 250
  obstruction of cystic duct, 252
Pericarditis in Bright's disease, 182
  purulent, complicating typhoid fever, 151
Pericardium, diseases of, 109
Peritoneum, carcinomatosis, miliary, of, 85, 221
Pernicious anemia, 173
Pneumothorax, with discovered perforation, 56
Poisoning by carbolic acid, 39
Polyp, carcinomatous, of cervix uteri, 307
Pons varolii, hemorrhage into, 300, and 305
Potts, C. S., discussion, 45
  tumor of spinal dura mater, 41
President's address, 1
Pylephlebitis, secondary to acute appendicitis, 233

RECTUM, carcinoma of, 85, 250
Riesman, David, anomaly of basilar artery, 64
  cancer of stomach, 29
  case of carcinoma of rectum, with miliary carcinosis of peritoneum, 85
  congenital hydronephrosis, 253
    inguinal hernia, 60
  discussion, 12, 56, 70, 78, 103, 147, 179, 182, 277, 303
  gangrene of lung, 63
  intestinal obstruction by peritonitic band, 302
  Meckel's diverticulum and omphalomesenteric duct, 193
  Melkinow-Raswedenkow method of preserving organs, 63
  primary cancer of gall-bladder; extensive secondary involvement, 9
  tumor of infundibulum, 64

Roberts, J. B., internal strangulation of small bowel by thread-like band, 35

SAILER, JOSEPH, abscess of lung, 39
  alterations in spinal cord in azoturia, 286
  calculus of spleen, 304
  changes in central nervous system after aseptic injury, 45
  congenital enteroptosis, 154
  contribution to pathology of choroid plexus, 273
  discussion, 5, 44, 63, 144, 153, 157, 226, 237, 295
  heart with two ventricular aneurysms, 21
  nucleolar degeneration of ganglion cells, 217
  renal cirrhosis, 105
  typhoid ulcer in appendix, 304
  and D. L. Edsall, peculiar bodies found in central nervous system, 96
Sarcoma, mast-cells in, 183
  melanotic, of liver, 33
  multiple, 105
Schmoerl's stereopticon views of heart-lesions, 184
Schreiner, E. R., gastric carcinoma, 40, 303
  discussion, 147
Scott, J. A., discussion, 179, 182
  specimens from case of pernicious anemia, 173
Smith, H., acute epiphysitis and purulent pericarditis complicating typhoid fever, 151
Spiller, W. G., on amyloid, colloid, hyaloid and granular bodies in central nervous system, 277
  discussion, 38, 44, 49, 78, 99, 167, 217
  sections from sternocleidomastoid muscle in case of congenital torticollis, 282
  tumor resembling in structure choroid plexus, 276
  and W. W. Keen, two cases of wrist drop from injury of musculo-spiral nerve from fracture of humerus, 100
Spinal cord in azoturia, 286
Spleen, calculus of, 304
  in leukæmia. 227
  rupture of, 181
Splenic capsule, involvement of, in colloid cyst, 104
Steele, J. D., discussion, 35, 103, 104, 240, 250
  echinococcus cyst of liver, 106
  review of literature of Koch's Tuberculin R., 49
  rupture of aorta, with flap-like dissection, 241
  and J. P. C. Griffith, cystic kidneys, 303
  and J. H. Musser, diseases of myocardium, 116
Stengel, Alfred, blood in diseases of cardio-vascular system, 134
  cirrhosis of liver, with jaundice, 69
  discussion, 32, 60, 71
  obstruction of common bile-duct and suppurative cholangitis, 72
Sternocleidomastoid muscle, sections from, in case of torticollis, 282
Stewart forceps, apparatus for suspending, 149
Stomach, carcinoma of, 29, 40, 62, 305
  hour-glass, 183
Strangulation, internal, of bowel, 35
Swan, J. M., discussion, 157, 273
  dissecting aneurysm of aorta, 40
  tuberculosis of kidney, 34
Symposium on pathology of diseases of cardio-vascular system, 109
Syphiloma of liver, 103

TAYLOR, A. E., aneurysm of arch of aorta; hour-glass stomach, 183
  discussion, 11, 33
  marked fibroid myocarditis, with extensive valvular contraction, 183
  sarcoma presenting many mast-cells, 183
  urine in diseases of cardio-vascular system, 138
Taylor, J. G., multiple abscesses of lung following pneumonia and empyema, ulcerative endocarditis, 245
  obliteration of orifice of right coronary artery, 243
Testicle, tuberculosis of, 267

Toluidin blue, microchemic reactions of, 208
Torticollis, sections from sterno-cleidomastoid muscle in, 282
Tubal gestation-sac, with unruptured membranes, 145
Tuberculin R., Koch's, review of literature of, 49
Tuberculosis of kidney, 34
  miliary, acute, 19
  of myocardium, 92
  of testicle, 267
Typhoid fever, acute epiphysitis and purulent pericarditis in, 151
  with irregular lesions, 157
Typhoid ulcer in appendix, 304
Tyson, James, discussion, 143

ULCERATIVE endocarditis, 245
Uremia, case of, 5
Ureter, obstruction of, 306
Urine in diseases of cardio-vascular system, 138
Uterus subseptus, 190

VASCULAR SYSTEM, diseases of, 109, 126

WADSWORTH, W. S., discussion, 167
  pathology of color perception, 161
Westcott, T. S., discussion, 303
Weil's disease, 65
White, F., discussion, 79
Williams, H. L., carcinomatous polyp of cervix uteri, 307
  case of extra-uterine pregnancy, 168
  chorio-epithelioma; deciduoma malignum, 79
  dermoid cyst obstructing labor and requiring Cæsarian section, 180
  discussion, 173, 273
  malignant adenoma of intestine, 91
  tubal gestation-sac, with unruptured membranes, 145
  uterus subseptus, 190
Wilson, J. C., diseases of endocardium, 122
Witmer, A. F., discussion, 5
  and S. M. Morton, tumor of spinal meninges, 2
Wrist drop, 100

288/10

41B
110